JOURNAL OF CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY

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The van Ophuijsen Memorial Volume

A Historic Perspective — *J. H. W. van Ophuijsen*
The Continuous Case Seminar — *H. L. Flowers*
Some Biochemical Considerations of Psychotic Behavior — *Hudson Hoagland*
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Experimental Abridgment of the Rorschach Test — *Max Valentine*

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Statement of Purposes

The JOURNAL OF CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY is dedicated to the search for the fundamental factors in the etiology and pathogenesis of psychiatric disorders; to the training of an alert, progressive, and qualified psychiatric personnel; and to the stimulation and support of all phases of psychiatric service and research—biologic, chemical, psychologic, physiologic, and social.

In the pursuit of these aims, the JOURNAL OF CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY will venture wherever the quest may lead. Its sole criterion will be the promise of an increment in the understanding of the mind's ills. It will seek, above all, to bridge national boundaries, language barriers, and the artificial demarcations of schools and trends.

The JOURNAL OF CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY holds that there is no justification for the present discrepancy between the rich scientific technology available to psychiatry and the poverty of techniques in current use in diagnosis, treatment, and research; no need for the gap between the promise of research and the paralysis of inquiry; no excuse for the lag of years between discovery and publication, demonstration and application in practice; no sound reason for the tragic chasm between the desire of the public for psychiatric education and guidance and the failure of the profession to provide the inspiration and leadership that would fully mobilize all latent potentialities. In brief, the JOURNAL OF CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY is dedicated to the fulfillment of psychiatry as a science and a humanity devoted to the interests of all mankind.

The JOURNAL OF CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY looks on psychiatry as an integral whole that is, in turn, an organic part of the world of science. It seeks to be not a mere sheaf of passive paper, but a dynamic organism by means of which its editorial board will endeavor to utilize every opportunity and facility in the field of human knowledge to fulfill the tasks to which it is dedicated.

JOURNAL OF CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY

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The van Ophuijsen Memorial Volume

VOLUME XII, NUMBER 2, APRIL-JUNE, 1951

The present volume of the *Journal of Clinical and Experimental Psychopathology* is dedicated to the memory of Dr. van Ophuijsen and will present, during the course of 1951, reports of researches which he initiated, in which he participated, or which were in the process of completion at the time of his death, May 31, 1950.

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"...we dedicate The Creedmoor Institute for Psychobiologic Studies and the lives and dreams of its staff to the hopes of all men and women—that they and their children may enjoy better health and live happier lives."

GENERAL CARLOS P. ROMULO

AT THE DEDICATION OF THE CREEDMOOR INSTITUTE FOR PSYCHOBIOLOGIC STUDIES

Dr. J. H. W. van Ophuijsen, Director of the Institute; General Carlos P. Romulo, at the time President of the United Nations General Assembly (Fourth Session); Dr. Harry A. La Burt, Senior Director of Creedmoor State Hospital, Queens, New York.

WHEREAS

Psychiatry has recently suffered a grievous loss through the death of the great Dutch-American psychiatric pioneer, disciple of Freud, brilliant psychiatric clinician and leader, Johan H. W. van Ophuijsen

IT IS PROPOSED

that the First International Congress of Psychiatry pay tribute to his efforts for the advancement of psychiatry as a humanity and as a science by a resolution of sorrow and homage.

—Resolution adopted unanimously in Paris, September 1950.

JOURNAL OF CLINICAL AND EXPERIMENTAL PSYCHOPATHOLOGY

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A Historic Perspective

(EXCERPTS FROM AN ADDRESS DELIVERED BY J. H. W. VAN OPHUIJSEN, M.D. ON THE OCCASION OF THE DEDICATION OF THE CREEDMOOR INSTITUTE FOR PSYCHOBIOLOGIC STUDIES, FEBRUARY 9, 1950)

In the many years during which I have practiced as a psychiatrist I have naturally had the opportunity of becoming acquainted with a great number of well-known people and of witnessing the new developments in our field. Even during my premedical years I became interested in my specialty, and I learned Freud's name through his translation of Bernheim's book on hypnotism. His articles written during his early years soon became known to me, and I was ready for psychoanalysis when destiny kept me in Zurich, instead of allowing me to settle down in Constantinople, as I had planned to do. In the meantime, however, my professor of psychiatry, Dr. Jelgersma, the well-known neurologist, had inspired me with a deep interest in clinical psychiatry and I believe that I owe it to him that I have always been able to find a stable equilibrium between clinical psychiatry and what is called depth psychology. My contact with men like Bleuler, Jung, Riklin, and Maeder contributed considerably to this equilibrium. That they were able to change so completely later on has remained an enigma to me, as has also the regrettable fact that two great men, Freud and Kraepelin, have never been able to find a common ground.

After these first years of initiation into the mysteries of psychoanalytic psychiatry, I witnessed the major developments in the field. When I came to Zurich in 1909 the interest in the word-association took its place. In the Freudian camp there were the developments of the libido theory and the investigation of the contents of the unconscious. Then followed the introduction of narcissism (1914), the reformulation of the internal conflict on the basis of the hypothetical death urge (1921), and a whole series of monographs that defined the repressing system, the so-called Superego, culminating in that charming little monograph, "Civilization and Its Discontents." Undoubtedly Freud would have attacked the problems of the Ego, had he lived longer.

On the clinical side I became acquainted with Pavlov's experiments on conditioned reflexes in his laboratory in St. Petersburg (Leningrad) in 1913, with Wagner Jauregg's fever therapy for general paresis, with a new development of occupational therapy which has not yet found its way into this country, with the shock therapies, during the war years with a re-emphasis on psychotherapy, including group therapy, psychoanalytically oriented or otherwise, with so-called psychosurgery, and finally, with the biochemical treatment of certain psychiatric conditions, especially depressions of various categories.

In looking back, I find a few memories that support my conviction that there is always a physical factor present in even the most purely "psychogenic" disorder, and certainly always in those disorders, the psychologic manifestations of which can be understood, even though they do not "explain" the morbid process. The first memory is connected with Jelgersma who in his inelegant manner of speaking used to say: "Schizophrenia is hysteria plus autointoxication." The second memory has Freud as the central figure. It was in the summer of 1927 that I spent a couple of weeks at a resort near Vienna where he stayed too. The well-known endocrinologist Steinach happened to be there and on one occasion when we talked about his work Freud said: "I am sure that within the near future all those disorders which we treat with psychoanalysis will be cured by endocrine treatment, but I am glad it is not yet that far, for it has given, and is giving us still the opportunity of studying the psychic mechanisms which we might not have found had we reached that stage earlier."

There is a faint recollection of experimental endocrine treatment of schizophrenics, I believe in the late twenties; finally the relatively new experience of becoming acquainted with the nosological entity identified by Schilder and Bender as childhood schizophrenia. Its characteristic disturbances in practically all areas of functioning—more severe in one area, less severe in another, thus accounting for the uneven development of the child—can be explained only on the basis of an autointoxication in which endocrine imbalance is involved.

As you see, I was prepared to become interested in any kind of research that made this problem of autointoxication and its causes its leading theme. As luck would have it, I found the Drs. Sackler and those of you who know their work will understand why I threw in my lot with them and decided to participate in their research carried out in our practice. I am convinced that we have found one entrance to the way of solving my problem. That our method has unexpected therapeutic value renders it the more attractive.

The Continuous Case Seminar

H. L. Flowers, M.D.

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The graduate training of residents in psychiatry presents many problems, but none are so difficult of solution as the problem of teaching therapy.

The embryo psychiatrist is at a complete loss when he first confronts a patient. Through his mind run many vague ideas about dynamics, transference, resistance, etc., and he is simultaneously eager and apprehensive. Frequently he is overwhelmed with anxiety when he confronts a tense patient, and if he does not secure prompt response to his psychotherapy he becomes discouraged. Of course, he is helped by his supervisor, but he still retains the impression that the inadequacy he feels is peculiar to himself.

When, however, a group of young therapists have a patient presented to all in common and the progress of the patient is checked by an older psychiatrist in whom they place confidence, they lose the feeling of isolation and can view their own therapy more objectively. The resident may, as it were, stand aloof and view himself as exemplified by his fellow resident, from a distance.

At the Veterans Administration Hospital, the Bronx, Dr. van Ophuijsen had developed the continuous case seminar to a high degree, and his seminars were always a source for fruitful discussion by the residents. Dr. van Ophuijsen had a very warm understanding of the needs of the young psychiatrist, and an uncanny intuition in sensing their difficulties.

The procedure of the continuous case seminar was as follows. One of the residents was asked to select a patient who was suffering from a psychoneurosis and who presented a good amount of psychopathology. The resident also tried to select a patient with an average or better than average intelligence, and one who was co-operative. In making his selection the resident was advised by Dr. van Ophuijsen, who occasionally would change the selection.

In the first seminar the resident acted as the patient and was interviewed by another resident. This interrogation was done just as though it were with the patient, though the patient himself never came before the conference. Dr. van Ophuijsen would frequently stop the interview and point out some error in the questioning or call attention to some evidence of psychopathology and discuss it with particular reference to the patient's general condition. It has been found that the resident group is quick to see errors in the interview technique of the presenting resident, even when they themselves make the same mistake. And not infrequently one of the residents will note with some confusion that he himself has had some difficulty, the cause of which he now understands.

Reviewed in the Veterans Administration and published with the approval of the Chief Medical Director. The statements and conclusions published by the author are a result of his own study and do not necessarily reflect the opinion or policy of the VA.

The differences between the diagnostic and the therapeutic interview were brought out and discussed. Sometimes, two sessions were devoted to history taking. The residents were encouraged to interrupt the interview and ask questions about the case, or the reason for some question. Dr. van Ophuijsen kept the seminars informal and everyone present felt at ease in bringing his own problems to the group. This informality is of considerable importance, for if the group becomes too large or too formal the individual residents tend to be less frank and will not bring their problems to the group. When the group is small and the atmosphere friendly and informal, the residents are eager to tell of their troubles and seek advice.

After the first one or two seminars devoted to diagnostic interviewing, the remaining sessions were spent in discussing and analyzing the therapy. The presenting resident was instructed to write down as much detail as possible of the separate therapeutic interviews with the patient. Occasionally the patient would resent notes being taken, in which case the resident would have to tax his memory to record the many details at the end of the interview. A recording device was used but usually it has been found that the patient was inhibited by the knowledge of being recorded, and his productions were limited.

In recording the productions of the patient, it is important for the resident to make full notes as to the mood of the patient. Any evidence of emotional response such as increased restlessness, flushing of the face, etc., should be recorded and a note made as to which questions elicited the response. It is also important for the resident to be frank in reporting any action or attitude of his own which may have been contributory to the patient's response. Sometimes it is more instructive to learn the thinking and emotional reactions of the resident than the psychopathology of the patient.

These interviews were read to the group in as much detail as possible. Here again Dr. van Ophuijsen would frequently interrupt in order to bring out some important point in dynamics or therapy, or to discuss some phase of the transference situation (and all too often the countertransference). Every so often some point would be raised that required extensive discussion. At such times Dr. van Ophuijsen would set aside the next session for the problem. He would then give a didactic discussion of the point raised, following which the meeting would be turned over to open discussion. This enabled the residents to clarify any questions or doubts they may have had.

Whenever there was any definite change in the patient's condition, the group would discuss what factors were most contributory to the change, and an attempt would be made to prognosticate as to future developments. An average case would be followed from 4 to 8 months, depending upon how much progress was made and how much material the patient produced.

It has been found that the residents were able to identify themselves with the presenting resident, yet could be sufficiently objective to perceive the errors or good elements of the therapy recorded. And frequently the various residents would quote from their own experiences and compare their therapeusis with the therapeusis of the presenter.

Dr. van Ophuijsen had an extreme sensitivity for the therapeutic situation, and it was his custom to spend considerable time inquiring into why certain questions were asked, and even why a question was phrased in a certain way. Thus it would come to light that the resident

had some preconceived notion about the patient and was trying to prove his point, rather than ascertain the actual truth.

In these sessions the resident group was encouraged to enter into the discussion and clarify their thinking, and no matter how simple the problem raised, Dr. van Ophuijsen would patiently discuss the matter thoroughly.

The continuous case seminar has further value in that the residents learn how slowly progress is made in some cases. Everyone interested in graduate psychiatric training knows how frequently residents become discouraged when their patient does not improve after a few treatments. But when they see that the case presented also progresses slowly in spite of receiving intensive therapy, the residents regain some of their self-confidence.

In summary, the continuous case seminar, as developed by Dr. van Ophuijsen, has been found extremely helpful in teaching young psychiatric residents technique in psychotherapy.

RESUMEN

En el seminario de casos clínicos de observación continuada que dirigía el Dr. Van Ophuijsen, el médico residente presentaba el caso con todo detalle e informaba semanalmente sobre las entrevistas sostenidas con el paciente. Se ha comprobado que dichas presentaciones tienen un gran valor en lo que se refiere a la instrucción de los médicos residentes sobre la técnica de la entrevista psiquiátrica y de la práctica de la psicoterapia. Los residentes que asistían al seminario pudieron apreciar objetivamente los defectos y errores, así como las ventajas del método psicoterápico empleado por el residente encargado de la presentación del caso y, al identificarse psicológicamente con él, pudieron emplear con facilidad su técnica ellos mismos. El propio Dr. Van Ophuijsen utilizó a veces los casos presentados para enseñar los fundamentos dinámicos y la aplicación práctica de la psicoterapia. Se comprobó frecuentemente que cualquier sistema empleado para registrar la entrevista, dificultaba la relación entre el médico y el paciente. El seminario de casos clínicos de observación continuada, demostró ser de gran valor en el adiestramiento de los médicos psiquiatras residentes.

RESUME

Dans le séminaire des cas suivis, tel que mis en œuvre par le Dr. Van Ophuijsen, l'interne a présenté le cas en détail et a fait toutes les semaines un rapport sur les diverses entrevues qu'il a eues avec le sujet. On a constaté que ces rapports présentés par les internes étaient très utiles pour enseigner aux internes la technique des entrevues psychiatriques et de la psychothérapie. Les internes du séminaire ont pu se rendre compte objectivement des divers défauts et erreurs, ainsi que des bonnes qualités de la psychothérapie adoptée par l'interne faisant rapport, et étant donné que les internes intéressés travaillaient dans le même cadre que l'interne faisant rapport, ils ont pu tirer un parti avantageux de ces leçons dans leurs travaux. Le Dr. Van Ophuijsen lui-même a utilisé la substance de ces rapports pour enseigner de temps en temps la théorie dynamique et la pratique psychothérapeutique. On a fréquemment observé que l'emploi d'instruments enregistreurs d'un genre ou d'un autre était nuisible à la prise de contact entre le docteur et son sujet. Le séminaire des cas suivis s'est montré d'une très grande utilité dans l'enseignement des internes psychiatres.

ZUSAMMENFASSUNG

In der von Dr. van Ophuijsen gehaltenen Art des fortlaufenden Studiums eines gegebenen Krankheitsfalls stellte ein Assistenzarzt den Fall ausführlich vor und berichtete jede Woche über die Konsultationen, die er mit dem Patienten gehabt hatte. Diese Art der Darstellung erwies ihren Wert als Lehrmaterial, um Assistenzärzte in psychiatrischen und psychotherapeutischen Methoden zu unterrichten. Die Teilnehmer waren in der Lage sowohl die verschiedenen Fehler als auch die Vorzüge in der Psychotherapie des vortragenden Assistenzarztes objektiv wahrzunehmen. Da die Zuhörer sich mit dem Vortragenden identifizierten, konnten sie das Erlernte ohne Schwierigkeiten selbst anwenden. Gelegentlich benützte Dr. van Ophuijsen selbst das Vortragsmaterial, um die Grundlagen seiner dynamischen Theorie und psychotherapeutischen Praxis zu lehren. Es ergab sich oft, dass die verschiedenen Arten der Aufzeichnungsapparaturen das Verhältnis zwischen Arzt und Patienten störten. Diese Art des fortlaufenden Studiums erwies sich als sehr wertvoll in der Ausbildung von psychiatrischen Assistenzärzten.

Some Biochemical Considerations of Psychotic Behavior

Hudson Hoagland, Ph.D.

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It is an article of faith of most students of biology that all human conduct, from the simplest reflex to the achievements of a Newton or a Shakespeare, depends upon the molecular dynamics of the nervous system.

Stanley Cobb[1] has pointed out that in present-day psychiatry the multiplicity of causes of psychopathological disturbances can be profitably expressed by the interplay of four main categories of events: (a) genogenic or hereditary causes, (b) histogenic causes or those related to visible lesion of the tissue, (c) chemogenic causes due to ultramicroscopical (molecular) changes in tissue function and structure and (d) psychogenic causes due to maladjustments in interpersonal relations. To quote Cobb: "I would insist that the old dichotomies 'functional or organic,' 'mental or physical,' are not only wrong but lead to bad habits of thinking because they lead to static and obsolete ideas and do not allow for modern pluralistic and dynamic ideas of matter and structure. Physiology is the basis of clinical diagnosis and no physiologist would accept for a moment the clinical jargon that uses the word 'functional' to denote 'psychogenic.' Anybody who stops to think realizes that no function is possible without an organ that is functioning and therefore no function takes place without structural change. Every symptom is both functional and organic. It is never a question of 'either—or.'"

Patients in mental hospitals suffering from dementia praecox (schizophrenia) and other so-called "functional" disorders have consistently failed to show post mortem histological changes that can be attributed to the psychosis per se. Present histological procedures cannot, of course, reflect the subtleties of chemical dynamics in the living tissues, so that these negative results have little bearing on the possible rôle of genetic and chemical factors that may be involved in the psychoses.

Without wishing in any way to disparage the importance of psychogenesis in the development of the psychoses, I submit that the persistent question arises as to why some persons become psychotic in the face of relatively unstressful social situations, while others do not, despite

Work on the adrenal cortex described in the second half on this paper has been helped by grants and contracts from the National Institute of Mental Health of the U. S. Public Health Service and by the Office of Naval Research. It has also been aided by grants from The Williams Waterman Fund of the Research Corporation, the G. D. Searle Company, the Scottish Rite Committee for Research on Dementia Praecox and by Mrs. Albert Lasker through the F. C. B. Foundation.

Parts of this paper have appeared in other publications by the author. However, in the following pages I have attempted to co-ordinate the results of several quite independent studies of the chemical physiology of mental patients in relation to the major psychoses.

great psychological stress. To say that early conditioning explains the difference is not to escape from the influence of the genogenic and chemogenic causes, since the conditioning of early experience must operate via the molecular organization of the nervous system which it in turn modifies. Mental disease as a form of behavior must ultimately be comprehensible in terms of its concomitant molecular physiology. A behaving disembodied psyche is a meaningless concept—the psychoses present an outstanding challenge in psychosomatic relations.

I should like now to consider certain ideas that shed some light on the chemical and genetic aspects of psychoses.

The structure and function of any living cell depends upon a dynamic steady state of energy transformations within the cell. Enzymes are organic catalysts. They are proteins combined ordinarily with specific molecular configurations called prosthetic groups. Enzymes control the breakdown of foodstuffs to simpler metabolic products and make available energy for the syntheses of cell constituents, for the liberation of heat, for the mechanical work of muscle and of excretory cells, and for the electrical events constituting nerve action currents. Thus in both the anaerobic or fermentative breakdown of a foodstuff and also in its oxidative breakdown a variety of cellular enzymes participate, each one promoting a specific and relatively simple chemical change. Glucose, for example, is broken down inside cells in some 30 organized sequential steps and cycles to simpler metabolites. The structure and normal excitability relations of the cells of the brain thus involve a continuous exchange of matter and energy between the cells and their environment. The electrical polarization and excitability of nerve tissue is a consequence of this exchange, and neurone excitability, the nerve message itself and its synaptic transmission can be profoundly modified by alterations in the chemical relations of the cell to its environment through substances that accelerate or inhibit the action of specific enzyme systems within the cell. Such chemical modifications of cellular physiology and of the messages upon which behavior depends are not necessarily accompanied by microscopic changes observable with present-day histological techniques.

Similar enzyme systems are found to be present very generally in muscle, brain, liver, spleen and glandular tissues. The steps of anaerobic fermentation and of aerobic oxidation in tissues from one organ to another and from one animal species to another are far more similar than they are different.

It is generally accepted[2] today that vitamins act essentially as catalysts—either as coenzymes or as prosthetic groups within protein enzyme molecules. This is certainly true of some of the B vitamins whose rôles in cellular metabolism are best understood. Thus diphosphopyridine nucleotide and thiamine are prosthetic groups associated with proteins that form enzymes involved in glycolysis. In like manner hormones probably exert their effects on their specific target organs by modifying the action of cellular enzyme constituents. Price, Cori and Colowick[3] have shown that the diabetogenic hormone from the anterior pituitary specifically inhibits hexokinase, an enzyme involved in the utilization of glucose by animal tissues, and that it does this both *in vitro* and *in vivo*. This inhibition is quantitatively counteracted by the hormone insulin.

For reviews of the influence of hormones on other enzymatic reaction see Jensen and Tennenbaum[4] and Hoagland.[2a]

ENZYME KINETICS AND PSYCHOTIC BEHAVIOR

Evidence reviewed by Hoskins in his 1945 Salmon Lectures[5] indicates that the metabolism of schizophrenic patients as a group displays abnormalities. Careful studies of basal metabolic rates of oxygen consumption of schizophrenics show that these rates are lower than those of normal persons by a small but statistically significant amount. MacFarland has pointed out similarities between the effects of anoxic anoxia on the behavior of normal men and the behavior characteristic of schizophrenic patients. Both the patients and anoxic normals display apathy, poor judgment, inappropriate affect, loss of self-control, emotional outbursts and tangential associations. Similar states, including hallucinations, may be produced in normal persons by histotoxic anoxia of the brain caused by the action of alcohol and other narcotic agents. Hoskins, in his review, cites the work of Looney as indicating a significantly greater amount (by some 50 per cent) of lactic acid in the blood of schizophrenics compared to normal persons, following bouts of exercise. Since blood lactic acid has been shown directly to reflect tissue lactic acid production, this indicates a deficiency in the metabolic events of the carbohydrate cycle of muscle responsible for the oxidative removal of lactic acid.

Schizophrenic patients display great tolerance to thyroid hormone. Cohen has kept some of his patients on doses of 28 grains of thyroid per day without ill effects. This is an amount which would produce enormous metabolic stimulation in normals. Thus thyroid, which usually greatly accelerates metabolism, does so much less effectively in schizophrenic patients. Other suggestive abnormalities of metabolism in schizophrenics are seen in their excessively high insulin tolerance and in a tendency to display diabetic type sugar tolerance curves. (See Hoskins[5] for citations.)

The evidence just reviewed suggests derangements of intermediate tissue metabolism in schizophrenic patients. Basal metabolic rate measurements and blood lactic acid measurements primarily reflect muscle metabolism. But evidence exists implicating derangements of brain metabolism in some psychotic patients. These derangements we would not necessarily expect to be reflected in the oxygen consumption of the brain, as measured by differences in oxygen content of arterial blood and of venous blood coming from the brain. The brain operates with a considerable factor of safety for both its oxygen and glucose supply. The limiting factors in tissue respiration are seldom the initial substrate concentrations but rather the extent to which the enzymes involved are capable of saturation with their respective intermediate substrates. Thus the overall oxygen consumption of the psychotic brain might be normal, but some one or more of the series of intermediate enzyme-catalyzed steps in the oxidation of glucose might be so altered as to produce effects that would modify cellular excitability and tissue function.

We have demonstrated[6, 7] that, with the advancement of spirochete infection of the brain in general paresis, first one and later another enzyme-catalyzed link in the oxidation of carbohydrate by the cerebral cortex becomes the limiting factor ("bottleneck"), or chemical pacemaker for cortical respiration. Accompanying these pacemaker shifts are advancing degrees of psychotic behavior. For example, our evidence from studies both *in vitro* and *in vivo* indicates that the cytochrome oxidase step in cortical respiration is the slowest one and hence the limiting chemical pacemaker in *advanced* general paresis. In *intermediate* states of paresis the succinodehydrogenase step appears to be the limiting factor while in *normal* persons and in *early* general paretics

a third unidentified enzyme system is the chemical pacemaker. As yet we have been unable to test this view with other types of psychotic patients, though the low brain iron of schizophrenics reported by Freeman[8] is at least consistent with the hypothesis that the iron-containing cytochrome system may be the limiting factor in brain respiration of this group.

In our studies of chemical pacemakers we showed by several procedures that *changes* in the frequencies of dominant cortical alpha brain-wave rhythms were proportional to *changes* in cortical respiration. In our series of experiments on chemical pacemakers we further found that when temperature (via diathermy) accelerates these rhythms their energies of activation, calculated by the Arrhenius equation, were identical with those obtained by studies of oxygen consumption in Warburg manometers for extracted specific respiratory enzymes, when these enzymes are the limiting factors for oxygen consumption. For a review of chemical pacemakers in relation to physiological rhythms see Hoagland.[9]

If brain enzyme kinetics are important in the psychoses, several rather interesting phenomena may receive interpretation. Inhibiting brain respiration by depriving it of its glucose fuel via insulin tends to relieve some psychotic patients, but, correspondingly, depriving patients of oxygen and thus slowing respiration has little if any therapeutic value. If toxic intermediate metabolites tend to accumulate in brain cells as a result of abnormal enzyme kinetics, depriving the cells of oxygen would do nothing to remove these intermediates. Oxygen may be thought of as a sort of locomotive pulling a train of hydrogen across a bridge of carriers—carriers which themselves depend on the normal dynamic balance of the enzyme-catalyzed events constituting cellular respiration. If we deprive the brain of glucose we would expect that toxic intermediaries would be burned up more rapidly than normally, since their rate of accumulation would be blocked.

Another point of interest is that certain substances now known to inhibit brain respiration temporarily relieve psychotic symptoms in patients. Thus the barbiturates inhibit brain respiration *in vitro* by blocking the oxidation of glucose, lactate and pyruvate.[10] These barbiturates, sodium amytal in particular, may strikingly, though transiently, relieve some psychotic symptoms. Again it has been observed that inhibition of oxidation produced by cyanide and also by excessive amounts of CO_2 temporarily relieves symptoms.[10] On the other hand, in normal persons some of these inhibitors produce confusional states bearing some similarity to psychotic episodes. Various amines are known to inhibit respiratory enzymes by interfering with the action of their protein moieties.[10] Thus mescaline, which is a trimethoxy-phenylethylamine, is notable as a producer of hallucinations.

One is tempted to speculate in relation to these phenomena to the effect that tampering with the rates of intermediate metabolic events in the normal brain may produce psychotic-like disturbances, while the same interference in the cellular dynamics of patients may facilitate the temporary re-establishment of more normal brain function. If in the psychotic brain, in the course of its functioning, we picture a slowing of one or more steps in respiration because of the inadequacy of some particular enzyme concentration, metabolites on the glucose side of this block would increase in concentration and might leak out of their loci to be abnormally metabolized. Metabolites on the oxygen side of the block, on the other hand, might be unduly reduced in concentration and energy release dependent upon their oxidation would decline. Thus toxic proteoses might be formed instead of normal protein molecules. The common metab-

olite pyruvate is toxic above low concentrations, as is the C_3 dicarboxylic acid, malonic acid, which very closely resembles the C_4 dicarboxylic hydrogen carrier, succinic acid. In this latter case it is known that malonic acid inhibits respiration by competing with it for the enzyme succinodehydrogenase.[11] Malonic acid and succinic acid are structurally very similar. A derangement of metabolism that produced even a small amount of the former might extensively modify brain function.

What part of the brain may be the most likely place to look for molecular derangement of function in psychotic patients? A considerable body of evidence reviewed by Gellhorn[12] especially implicates the hypothalamus. Schizophrenic patients display evidence of a relative lack of responsiveness of their sympathetico-adrenal systems and relative overactivity of their vagoinsulin systems. This, together with other evidence, leads to the view that the difficulty is inherent in the hypothalamic motor centers of the sympathetic branch of the automatic. Gellhorn holds the view that "since in normal individuals, man and animals, the sympathetico-adrenal stimulation predominates over that of the insulin system, it seems likely that the therapeutic effect of the shock therapies in schizophrenics is linked up with the stimulation of the sympathetico-adrenal system," and he presents experimental evidence in support of this view. Gellhorn's hypothesis is in no way inconsistent with the metabolic views expressed above. It is hard to conceive of how reactivity could be permanently enhanced in a center such as the hypothalamus without acting through the enzyme systems involved.

While in our study of activation energies of brain waves in man we recorded cortical electroencephalograms, there is evidence from several sources indicating that the circuits conducting these waves pass through the hypothalamus and that the hypothalamus may actually be the rate-determining center for the waves.[13, 14, 15, 16, 17]

THE ADRENAL CORTEX AND ITS RELATION TO THE PSYCHOSES

In recent years investigators at the Worcester Foundation have demonstrated that there are marked deficiencies in responses of the adrenal cortex to stress in schizophrenic patients, when the patients are compared with normal controls and with psychoneurotic patients subjected to the same experimental stresses.

Unlike the adrenal medulla, the adrenal cortex has no nerve supply and its output of steroid hormones is entirely regulated by an anterior pituitary protein hormone, adrenocorticotrophin (ACTH). The release of ACTH is under the control of the hypothalamus by mechanisms that are still obscure. The adrenal steroids have far-reaching metabolic repercussions in the body. Recent studies from our laboratories indicate that 15 hormones are released into the blood stream in response to ACTH.[18] These hormones include cortisone, corticosterone, 17-hydroxycorticosterone, 11-desoxycorticosterone and 11-dehydrocorticosterone. Some of the adrenal steroids are regulators of salt and water balance (the desoxycorticosterone type of substances) and others, with oxygen or hydroxyl at the 11 and 17 positions of the steroid skeleton, are primarily concerned with protein, fat and carbohydrate metabolism.

Since 1940 we have been engaged in the study of adrenal responses to a wide variety of

workaday stresses in normal men and women and in those hospitalized for severe personality disorders. Our stresses have been of a physical, chemical and psychological character. In the early period of this work, changes in the urinary excretion of 17-ketosteroids constituted the only practical index of adrenocortical function in man. Later, a method of determining changes in the number of circulating lymphocytes became available, and more recently relative changes in seven urinary and blood indices have been used routinely by us in our adrenal studies. Thus increases in urinary 17-ketosteroids, corticoids (neutral reducing lipids), potassium, sodium, and uric acid, together with decreases in circulating lymphocytes and eosinophils, reflect enhanced activity of the adrenal cortex. Lymphoid tissue and other protein reservoirs are acted upon by the 11-oxysteroids which convert protein to sugar which in turn is stored in the liver. Uric acid excretion, lymphopenia and eosinopenia reflect this conversion. The excretion of potassium and sodium is affected by several adrenal steroids through the modification of kidney thresholds and changes in tissue electrolyte balance.

We have studied adrenal stress responses in approximately 200 normal persons. Our stresses have included exposures to cold,[19] and to heat and high humidity,[20] the prolonged operation of an airplane-type pursuit meter which quantitatively measures fatigability,[21] 208 test flights of 23 professional airplane pilots,[21] and the taking of examinations and interviews and of an especially designed frustration test (targetball test).[22, 23] We have extensively examined the pituitary-adrenal response to the chemical stress of ingesting large quantities of sugar.[22, 24, 25, 26, 27] In most of these tests urine and, usually, blood samples have been taken before each stress, 15 minutes after the end of the stress (usually lasting an hour) and again two hours later. The stresses, while varying in effectiveness, enhanced the adrenal activity of our normal population, as reflected in poststress percentage changes from pretest levels of the various urinary and blood indices, and we were able to correlate measured aspects of psychomotor fatigability with levels of adrenocortical response to the same stress in different individuals. These results were especially striking with subjects performing on the pursuit meter.[21, 28]

As a result of these findings we attempted to improve prolonged psychomotor performance by the administration of certain steroid substances and found that the synthetic steroid $\Delta 5$-pregnenolone, taken by mouth in 50-100 mg. doses significantly improved prolonged psychomotor performance, compared to what was observed after the taking of placebos.[29, 30, 31] This substance does not produce untoward side effects. Recent investigations in our laboratory have shown that this substance is converted by the adrenal cortex to adrenal cortical hormones.[18]

Schizophrenic patients as a group have failed to meet the daily stresses of living and have developed bizarre forms of behavior, necessitating hospitalization. We were interested to find that these patients, in general, displayed abnormal and inadequate adrenal stress responses as compared to controls, though the resting, fasting values of the above urinary measures, reflecting adrenal function, differed little from those of normal persons. We have studied approximately 100 chronic male schizophrenic patients in many of the same stressful tests we used with our control group.[19, 20, 21, 22, 24, 25, 26, 27, 28, 32, 33]

The target organs responding to adrenal steroids were normally responsive in our patients, as was demonstrated in control and patient groups by the injection of standard doses of Upjohn's lipo-adrenal extract. The response of the adrenal cortex to ACTH was, however, abnormally

sluggish in the patients, thus indicating that the deficiency lies at the level of the adrenal cortex itself. A total response index (abbreviated TRI) has been devised[22, 28] as a measure of adrenal responsivity to our stresses and to hormone injections. This consists of a mean of the sum of the percentage changes from prestress or preinjection levels of the two poststress samples of urinary sodium, potassium, uric acid, 17-ketosteroids, and cortins, plus twice the poststress percentage drop in lymphocytes added as a positive number. For example, all of 25 normal control subjects showed a TRI value of 20 units or greater following 25 mg. injections of ACTH, while only 28 per cent of 25 of our schizophrenic patients showed a response of 20 units or greater. Approximately this same ratio of normal responders to schizophrenic responders was found for all of our various stress tests.

In all of our schizophrenic group, including those who gave normal TRI responses (approximately one-third of the total), there was evidence of qualitative abnormalities in steroid metabolism.[28] Thus all the chronic schizophrenics as a group displayed on the average abnormalities of adrenal stress responses which appear to lie specifically at the level of the ability of the adrenal cortex to respond either to endogenous or to exogenous ACTH.

The abnormal responses were not corrected by two weeks of a vitamin- and protein-rich diet.[34] They were not found in hospitalized psychoneurotic patients who gave normal responses to 25 mg. injections of ACTH.[28]

The refractoriness of the adrenals to ACTH in the psychotic patients displaying this phenomenon was not absolute. Thus larger quantities of ACTH (75 to 100 mg.) produced TRI responses approaching that of the controls given 25 mg. doses, but qualitative abnormalities of response were even more in evidence.[28, 33]

We have found a direct relation between responsivity of the adrenal cortex to ACTH and prognosis with subsequent electroshock therapy (EST) in schizophrenia.[35] Thus the more normally responsive the adrenal cortex to preshock injections of test doses of 25 mg. of ACTH, the better the prognosis for subsequent shock therapy. Psychiatric improvement with EST, as measured on a well validated rating scale,[36, 37] gave a positive rank-order correlation of 0.78, $P < 0.01$, with pretreatment values of the TRI.

We have reported that normal persons show an increase of 50 ± 14 per cent in output of 17-ketosteroids in the first two hours after rising from a night's sleep.[19, 20, 22] This reflects a special form of adrenal stress response associated with waking and starting the day's activities. In general, schizophrenic patients display, as a group, a somewhat lower diurnal rhythm of excretion than do normal controls.

In 10 female patients suffering from involutional depression we also found evidence of adrenal cortical unresponsivity.[38] These patients averaged an insignificant (3 per cent) morning increase in 17-ketosteroid excretion over the night level, before they were given a course of electroshock treatments. During the treatment course the morning increase in output rose to an average of 32 per cent over the night level, and it was 25 per cent over the night level a month after the treatments had ended. Eight of the 10 women showed good social remission resulting from the treatments. Thus we see that alterations in adrenal responsivity to stress are found in psychotics other than schizophrenics and that the condition may be improved with therapy.

DISCUSSION

The adrenal abnormalities in psychotic patients may or may not be causal factors in the psychosis. Our data do not answer this question. It is possible that the adrenal deficiencies may precede the psychosis and may make for vulnerability to psychosis in the face of life's stresses. Kallmann[39] has shown by studies of a large number of schizophrenic patients and their families, including many pairs of homozygous and heterozygous twins, that there is undoubtedly a strong genetic component in this disease and it would be interesting to know if this component is associated with the adrenal abnormalities we have encountered in our patients. It is interesting in this connection that the more normal a patient's adrenals, the better his prognosis with EST.

It is perhaps equally probable that the adrenal aberrations accompany the development of psychosis or result from it. It is conceivable that prolonged overactivity of the hypothalamus may produce excessive release of ACTH and that this results finally in an adrenal refractory to it—an adrenal that responds sluggishly, both to endogenous and to exogenous ACTH, and one with an abnormal steroid output. If future work indicates that this is the case, we must look to the vulnerability of the central nervous system rather than to aberrations of adrenal mechanisms as the primary cause of psychotic behavior. The fact that neither injected cortisone nor ACTH is therapeutically beneficial in schizophrenia does not resolve the dilemma in favor of either of these hypotheses, though superficially it might appear to favor the latter one. Cortisone is only one of 15 adrenal cortical hormones and we have found that injections of 100 mg. of ACTH, while enhancing the total response, only accentuate abnormalities of adrenal response in our schizophrenic patients. Steroid hormones modify brain chemistry and physiology[2a] and thus might affect association processes and affective mechanisms. In this way adrenal deficiencies developing with the psychosis might modify brain mechanisms so as to make the psychosis chronic and refractory to therapy.

Thorn has considered nonpsychotic psychosomatic disorders in relation to ACTH and the adrenal cortex, and the following quotation from him is of interest.[40] "Studies with ACTH have also thrown light on the field of psychosomatic medicine. Since observing the fact that ACTH will cause a prompt and significant fall in circulating eosinophils in patients with intact adrenals, and that this fall is correlated with the outpouring of the so-called 11, 17-oxysteroids, we have studied the response of many patients and normal subjects to emotional as well as to physical stress. A period of anxiety may induce a fall in circulating eosinophils, thus indicating the release of a quantity of adrenal cortical hormone similar to that observed following the injection in the same individual of 25 mg. of ACTH. We believe that anxiety and emotional disturbance may result in stimulation of the hypothalamus, which in turn stimulates the pituitary and hence the adrenal cortex to liberate its steroid compounds. Knowing the widespread effects of ACTH on a variety of disease processes, we can visualize for the first time one pathway by which emotional and psychological factors may alter directly the state of inflammation in a joint, such as occurs in rheumatoid arthritis, or the course of rheumatic fever, or the course of peptic ulcer by modifying the secretion of gastric pepsin. In other words, every patient with an intact hypothalamic-pituitary-adrenal system has a potential mechanism within his own body

for suddenly modifying the course of a disease; and the importance of psychological factors in materially affecting the course of a systemic disease is beginning to be appreciated."

It is interesting that schizophrenic patients rarely display psychosomatic disorders such as arthritis and asthma—conditions promptly though transiently relieved by cortisone and ACTH. It is possible that this may be related to the abnormalities of steroid metabolism we have encountered in schizophrenic patients. This matter is now undergoing study.

We began this discussion by considering four causal factors that Cobb has suggested are useful in relation to the psychoses: genogenic, histogenic, chemogenic, and psychogenic factors. The studies of Beadle and his collaborators (see [41] and [42] for reviews), while apparently remote from psychiatry, seem to me to offer stimulating leads in relation to the first of these and its bearing on the chemogenic considerations reviewed above. This work consisted of genetic studies of the bread mold *Neurospora crassa*. Beadle demonstrated that the ability of this mold to synthesize each amino acid and each vitamin depends on a particular gene unique for each substance and he was able to locate the position of the genes for these specific synthetic processes in the chromosomes. Beadle showed that the enzymes necessary for the specific syntheses are produced by the genes. The genes can be inactivated by neutron and x-ray bombardment, permitting the deficiencies that are due to their elimination to be tested. Genic deficiencies thus produce specific losses in enzyme function.

Beadle,[41] in discussing the general implications of his work for human genetics, cites the incidence of phenylketonuria in man as an example of genetic deficiency. "Phenylketonuria," he writes, "is a recessive trait, although there is some evidence that carriers of it—individuals with one member of the relevant gene pair normal and the other defective—are not completely normal. Metabolically, phenylketonurics are characterized by inability to oxidize phenylpyruvic acid (a phenyl-keto acid) to the corresponding *para*-hydroxyphenylpyruvic acid. Consequently phenylpyruvic acid accumulates and is excreted in the urine. Individuals who excrete this substance rather than breaking it down are invariably mentally defective. They are usually classified as idiots or imbeciles. It is evident that the ability to oxidize phenylpyruvic acid is necessary for normal functioning of the central nervous system. Whether the relation between the chemical reaction and rational thinking is direct or indirect we do not know. It may be that accumulated phenylpyruvic acid is harmful. On the other hand, the reaction per se may be essential for the higher orders of thought."

The work of Kallmann previously referred to indicates a genetic factor predisposing to schizophrenia. The fact that epileptiform electroencephalograms are inherited, and with them accompanying tendencies to epilepsy, has been established by Lennox, Gibbs and Gibbs.[43] The most probable mechanism of operation of such genetic factors is through the control of enzyme syntheses, as Beadle has shown so beautifully for *Neurospora*. The great difference in the vulnerability of individuals to the role of psychogenic and social factors in producing psychoses may thus reside in the inadequate production of specific enzymes in important regions of the central nervous system and/or in association with adrenal cortical action. This matter calls for intensive investigation.

The quantitative assay of human brain enzymes, especially in an inaccessible region such as the hypothalamus, presents great difficulties. Fresh brain autopsy tissue is required from con-

trols and from psychotic patients of comparable age who have died from nondegenerative diseases. The study of the possible therapeutic benefit of enzyme material, administered to psychotic patients, is also not promising at present, because of the difficulty of obtaining adequate amounts of the numerous possible enzymes for clinical tests, their probable inactivation in the liver and their failure to pass the blood-brain barrier. However, in this connection one should recall that the severe psychosis associated with pellagra results from avitaminosis and can be relieved by the administration of nicotinic acid amide which is part of Warburg's coenzyme II. This substance plays an essential catalytic rôle in the oxidation of glucose. As knowledge of enzymes advances, one may hope that the administration of suitable ratios of amino acids and prosthetic groups may enable the possibly enzyme-deficient cells to construct suitable enzymes, though this would be unlikely if the deficiency were due to a genetic deficit.

Much research needs doing to test the hypothesis that psychoses are deficiency diseases. But the recent rapid advances of our knowledge of intermediate metabolism, and the application of some of this knowledge in the field of chemotherapy and of vitamin and endocrine disorders offers encouragement to those concerned with the problems of mental illness.

SUMMARY

This paper discusses experiments indicating that disturbances in the balance of enzyme kinetics may be associated with psychotic behavior.

Aberrations of adrenal steroid metabolism in schizophrenic patients are reviewed and considered in relation to possible etiological factors.

RESUMEN

El autor revisa su trabajo acerca de los "marcapasos" enzimáticos correspondientes a los ritmos de las ondas cerebrales alfa en personas normales y en psicóticos que sufren de paresia general; también trata otros trastornos del metabolismo hidrocarbonado en las principales psicosis. El trabajo considera los posibles efectos de ciertas anomalías en la dinámica de las enzimas y la determinación de las enzimas celulares por los genes en sus relaciones con los factores genéticos relacionados con ciertos tipos de psicopatología.

El artículo se refiere también a los estudios hechos por el grupo de la Worcester Foundation acerca de las respuestas obtenidas en individuos normales o esquizofrénicos con "stress" adreno-cortical y comenta las posibles relaciones que existen entre dichas perturbaciones endocrinas por una parte y la naturaleza de las enzimas y los factores genéticos correspondientes por otra.—(43 referencias).

RESUME

L'auteur rend compte de ses travaux sur les "entraîneurs" d'enzymes pour les rythmes d'ondes alpha du cerveau chez les sujets normaux et chez les sujets psychotiques souffrant de parésie générale, et examine d'autres aspects du dérangement de métabolisme relatif aux hydrates de carbone chez les principaux psychoses. Les effets éventuels d'anormalités de cinétique des enzymes et la détermination des enzymes cellulaires par les facteurs d'hérédité sont étudiés par rapport aux facteurs génétiques associés à certains types de psycho-pathologie.

Le mémoire donne un compte-rendu des études faites par le groupe de la Worcester Foundation concernant les réactions de tensions corticales-surrénales chez les sujets normaux et chez les sujets schizophrènes, et traite des relations éventuelles de ces dérangements d'endocrines chez les sujets malades par rapport à la nature de la schizophrénie, ainsi que par rapport aux facteurs génétiques et d'enzymes qui peuvent être en jeu chez ces cas.

ZUSAMMENFASSUNG

Der Autor gibt eine übersicht seiner Studien über Enzym-Schrittmacher für die Rythmen der Alpha-Wellen des Gehirns in normalen Personen und in an progressiver Paralyse leidenden Psychotikern und betrachtet auch andere Aspekte von Störungen des Kohlehydrat-Metabolismus in den wichtigeren Psychosen. Die etwaigen Effekte von Anormalitäten der Enzym-Kinetik und die Bestimmung von Zellenenzymen durch Gene findet Beachtung im Zusammenhang mit den genetischen Faktoren, die mit gewissen Abarten der Psychopathologie verbunden sind.

Die Schrift gibt auch einen überblick über die von der Forschungsgruppe an der Worcester Foundation angestellten Studien über die Reaktionen von normalen Personen und Schizophrenikern auf Belastungen der Nebennierenrinde und befasst sich mit etwaigen Zusammenhängen zwischen diesen Störungen der inneren Sekretion, dem Wesen der Schizophrenie und den eventuell implizierten Enzymen und genetischen Faktoren.

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Effects of Factors That Modify Cerebral Blood Flow on Hallucinations in Schizophrenia

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The nature of the factors that influence the occurrence of hallucinations in patients with various types of psychosis has been the object of speculation for millennia. In recent years psychogenic factors have been emphasized as the cause of hallucinations in schizophrenia. During the course of recent physiologic studies made here on patients with schizophrenia, hallucinations were observed to appear and disappear in some patients under the influence of factors that changed blood flow in the brain. The present discussion records the findings in the two patients, of the 30 studied, whose hallucinations showed the most marked changes during the study.

Case 1. *Temporary disappearance of hallucinations during inhalation of carbon dioxide:* M. R. K., a single, white, native-born woman, first entered the McLean Hospital in August 1927 at the age of 20, with a history of periods of apathy alternating with brief destructive rages, occurring during the three months preceding admission. The family history included many instances of mental aberration: (1) a nervous and excitable paternal grandfather, given to destructive rages, (2) an excitable, idiosyncratic maternal grandfather, who wore his hair long and dressed queerly, (3) a paternal great-aunt who became depressed in middle life and died in a hospital for mental diseases, (4) an excitable mother who had frequent temper tantrums, and (5) a father with manic-depressive psychosis requiring several admissions to hospitals. The patient's past history revealed measles, scarlet fever and influenza as a child. Catamenia, which began at the age of 15, was always irregular and accompanied by severe discomfort. A good deal of abdominal gas, diagnosed as caused by nervousness, had been present for several years before admission.

The patient had been a mild, amiable, affectionate child who grew and developed normally. However, terrifying nightmares occurred frequently during her childhood and at times she showed intense jealousy of her parent's friendliness toward or affection for other people. It was noted also during her childhood that daydreaming was frequent. She was found to have musical talent; during her studies she exhibited a phenomenal memory for musical scores. She showed no interest in boys, although her social life was otherwise normal. Before a recital she was to give at the age of 20, while a student at a famous conservatory of music, she began to complain of a feeling of pressure in the head and subsequently became irritable and fearful; she complained also of bad smells in the air and a brassy taste in the mouth. After a few weeks she began to feel that people were making derogatory remarks about her. Shortly before admission to the hospital, she attacked her mother and an aunt for having "stolen her beauty"; she was remorseful after this event. Preoccupied apathy, alternating with tantrums, was observed during the entire period and, in addition, odd poses and queer movements of the arms were noted from time to time. In the hospital she was untidy and mute for the most part, although she became agitated and explosively assaultive before her menstrual periods; hallucinations were present much of the time. Physical examination showed normal findings at all times and repeated hematologic, blood chemical and urinary studies showed no abnormalities; X-ray studies revealed atony of the colon. After six months she left the hospital improved, re-entering four times thereafter, each time with complaints similar to those at her first admission. Transitory marked improvement occurred spontaneously or under the influence of insulin or electroshock therapy during the first four hospital stays. The clinical diagnosis was schizophrenia.

During her most recent stay she showed evidence of hallucinations during all of her waking hours. She slept irregularly, for two or three hours at a time, and for the rest of the day listened and talked continuously to voices that seemed to come from above and usually to the left of her head. The comments of these voices were

not known, but judging from the patient's responses they referred usually to other persons and occasionally to the patient herself. The comments usually appeared to be scandalous in nature and, for the most part, were amusing or mildly shocking to the patient, judging from her expression and responses; on occasion they seemed to be terrifying. The present study was made five years after she had been admitted for the fifth time, two years after a course of electroshock therapy consisting of 6 treatments.

OBSERVATIONS DURING INHALATION OF CARBON DIOXIDE

On six occasions curare, 0.85 units per pound of body weight, was given intravenously, the injection lasting for two minutes. Immediate disappearance of the cough and gag reflexes occurred and respiration ceased two or three minutes later. As soon as coughing and gagging became impossible and before respiration ceased, a Magill catheter was inserted into the trachea at a level estimated to be 3 or 4 cm. above the carina. A mixture of 5 per cent carbon dioxide and 95 per cent oxygen was administered through the catheter at a rate of 5 to 6 liters per minute. Complete apnea lasting for 10 to 20 minutes ensued, with occasional hiccoughing. During the next 10 to 30 minutes the hiccoughs deepened and became convulsive gasping breaths. Efficient breathing began 30 to 60 minutes after the injection of curare. The cough and gag reflexes returned 15 to 30 minutes later, at which time administration of the carbon dioxide-oxygen mixture was terminated. The duration of administration was 45 to 90 minutes. Studies of the blood gases were made by the method of Van Slyke and Neill[1] and of the pH by means of a glass electrode.

The arterial blood showed the following changes:

	Oxygen Content	Carbon Dioxide Content	pH
Before inhalation	18.85 - 20.16	43.30 - 45.20	7.40 - 7.42
During inhalation	19.45 - 20.95	48.70 - 53.80	6.91 - 7.03

On the occasion of the first experiment, when the intratracheal catheter was removed, the patient looked directly at the interviewer, with a normal, mildly surprised expression on her face. When asked whether she heard any voices she replied, somewhat scornfully, "Well, I hear yours." Normal conversation relating to ordinary everyday topics continued for a few minutes, after which it was noted that the patient's eyes began to wander upward and to her left. Her responses to the interviewer's comments became faltering and her expression appeared puzzled. When she was asked whether she heard voices other than that of the interviewer she replied hesitantly, "Not very well." A minute or two later she reverted to her usual hallucinated condition, out of contact with her real surroundings. Three days later the experiment was repeated, again with disappearance of hallucinations for a brief period. On the next four occasions, however, no mental changes occurred as a consequence of the inhalation, even though the changes in the blood were the same as in the first two experiments.

Case 2. *Temporary reappearance of hallucinations under the influence of cerebral ischemia:* H. H. K., a married, white, native-born woman, entered the hospital in March 1947, at the age of 59, after having attempted suicide. Little was ascertained concerning the family history except for the fact that the father, an owner of race horses, spent much of his time in drinking and dissipation. The patient's early history was not known in detail. She had always been sensitive, shy, and rather withdrawn, and had disliked meeting people. After a sheltered childhood and some years at an expensive private school, she married at the age of 18; a son

was born to the patient when she was 19, after a long and difficult labor. Her husband drank to excess and took a mistress, whereupon the patient divorced him when she was 21. She remarried at the age of 32. In the interval, between the ages of 21 and 32, she managed her widowed father's household, traveling from one racetrack to another. Thirteen years before admission the patient went through some sort of mental upset characterized by severe depression. Five years before admission, after her husband entered military service, the patient became depressed, seclusive and distraught with worry. She felt also that people disliked her and made uncomplimentary remarks about her. The patient developed severe constipation, requiring massive doses of laxatives and many enemas; X-ray studies showed no cause for this condition. After three years in this condition she began to have terrifying visual and auditory hallucinations. At one point she claimed to be totally blind for approximately a week. Two months after the onset of hallucinations the patient's condition improved, and she was apparently normal for six months. Suddenly one day she became mute and was unresponsive for a few hours, after which she reverted to her former paranoid and hallucinated condition. She became seclusive again; she believed that her son was dead, that her husband was acting strangely, that people were discussing her evil character and that government agents were pursuing her. After six months of these beliefs she slashed her wrists, without doing much damage.

Physical examination on admission was not remarkable except for cuts on the wrists. Hematologic, chemical and urinary examinations showed nothing abnormal. The basal metabolic rate was minus six per cent. Electrocardiogram and electroencephalogram were normal.

On examination the patient was confused as to time and place, moderately depressed, and complaining of giving off a bad odor and of affecting other people adversely in some undefined way; she was greatly troubled by whispering voices that accused her of having syphilis and of having caused harm to her husband and son. She refused medications, believing them to be poisoned. A short time after admission she became mute and unresponsive for an hour and a half, during which time she grimaced continuously. She remained depressed, preoccupied and troubled by auditory hallucinations for several weeks. At times she had terrifying visual hallucinations that made her scream for minutes at a time. She was given nine electroshock treatments and seemed to have recovered from her psychosis; remission lasted for only two weeks and nine more treatments were given, again with benefit. Three weeks later relapse again occurred and a third course of nine electroshocks was given. She seemed well for 10 days and then once more became apathetic, seclusive and paranoid. The previously experienced auditory hallucinations recurred, but there was no return of her terrifying visual hallucinations. The clinical diagnosis was schizophrenia, paranoid type.

OBSERVATIONS OF PATIENT ON TILT TABLE

Eleven observations were made with the use of a tilt table so constructed that the patient was maintained upright at an angle of 60 degrees with her legs hanging free. Sodium nitrite in a dose of 0.2 to 0.4 grams was given orally 20 minutes before the start of each experiment. The systolic blood pressure with the patient in the flat position was between 114 and 140 mm. of mercury; it fell immediately after the patient was tilted up, the amount of decrease ranging between 3 and 30 mm. of mercury; the blood pressure then remained at about the same level for between 10 and 21 minutes thereafter. Subsequently it fell rapidly to or toward zero, returning to the control level as soon as the patient was restored to the flat position.

During the period of each experiment immediately after the patient was tilted to the upright position, when the blood pressure remained at a fairly constant level, the patient exhibited facial pallor and an increase in pulse and respiratory rate. Two to six minutes after being tilted to the upright position, accusatory, whispering, auditory hallucinations developed, or, if already present, became more marked. Several minutes later terrifying visual hallucinations appeared. These consisted of a vision of the shore of an island off the coast of Maine, at dusk, with

several men standing in a circular group watching intently while the patient's husband, in the center of the group, stabbed and dismembered the son born of her earlier marriage. She did not see her husband and son in this vision, but knew that they were there; she did see blood running over the sand out into the waves. Whispering voices accused her of being responsible for this event. This visual hallucination was the same as the one that occurred most frequently prior to her first shock treatments. Within two minutes after the patient was returned to the recumbent position all hallucinations disappeared and the patient laughed sheepishly or apologized for having made a scene, exclaiming at the silliness of her ideas. If tilted to the head-up position again, however, she once more experienced the terrifying visual hallucinations as a real sight; appreciation of their hallucinatory character returned when the patient was tilted back to the recumbent position.

DISCUSSION

In 1929 Loevenhart, Lorenz and Waters[2] reported that inhalation of carbon dioxide by mute or stuporous psychotic patients caused temporary restoration of these patients to or toward normal. Observations made here in such patients corroborate the observations in this regard of Loevenhart et al.[2] and other workers. The finding of a temporary disappearance of hallucinations in schizophrenic patients during inhalation of carbon dioxide may represent a phenomenon of similar mechanism. The rapid loss of effectiveness of carbon dioxide in this regard is to be expected on the basis of what is known regarding the development of tolerance to that substance.[3, 4]

Occurrence of temporary improvement in various psychotic manifestations after inhalation of carbon dioxide raises the question as to whether repetition of this procedure might effect lasting benefit. Observations were made here to study this matter; two patients with schizophrenia and one with depression were given inhalations as described above on from six to eleven occasions each, with no improvement which lasted for more than 15 minutes in any case. Subsequently they were given courses of electroshock therapy; only one, the patient described above, failed to improve. Indeed, one patient, a woman aged 79, diagnosed as suffering from manic-depressive psychosis, depressed phase, was given seven inhalations lasting from 26 to 48 minutes without effect on the psychosis, but when 6 electroshocks were given several weeks later there was complete remission of the psychosis. Studies made during one of the inhalation treatments in this patient showed the following changes in arterial blood:

	CO_2 Content	pH
Before CO_2	44.99 vol. %	7.40
During CO_2	58.60 vol. %	6.81

It is evident not only that carbon dioxide inhalations are of no lasting benefit in psychosis but also that the beneficial effects of electroshock cannot be ascribed to the carbon dioxide acidosis known to occur during such treatments.[5, 6]

The mechanism underlying the disappearance of hallucinations in schizophrenia under the influence of carbon dioxide cannot be characterized precisely because of the large number of factors that might be involved. Although the findings might possibly be explained on the basis of improvement in disturbed cerebral function owing to the well-known increase in cerebral

blood flow effected by increases in blood carbon dioxide content,[7, 8, 9, 10, 11, 12] specific effects of carbon dioxide and of acidosis on metabolism cannot be ruled out. Interpretation of the results of the experiments made with the tilt table is less uncertain: the reappearance of hallucinations was consequent to the changes in cerebral function that resulted from the decrease in cerebral blood flow known to occur under those circumstances.[13] In the patient described in detail in the present report (Case 2), full insight was evident when she was in the flat position; when she was tilted up, however, cerebral blood flow and insight diminished, the former rapidly and moderately and the latter slowly and completely. In addition to hallucinations produced or exaggerated when some of the patients were tilted up, changes in mood also were noted in the same or in other subjects.

Two subjects, one a normal control and one a chronically ill schizophrenic patient who had never had hallucinations, experienced vivid, brief visual images while under observation on the tilt table, immediately before the occurrence of syncope; these were memories of real events and their nature appears to be similar to that of the phenomena described by Penfield[14] as occurring with localized stimulation of the normal cerebral cortex. That the visual hallucinations that were experienced by Patient 2 while on the tilt table might be similar in nature is suggested by their repetitive occurrence, at first spontaneously during the illness and, after partial remission, during the experiments described above. The hallucination consisted in part of memories of real things, such as the site of the vision, a locality well known to the patient. Whether the hallucination was a memory of a psychotic misinterpretation of a real event or was a new formation consisting of memory combined with or changed by imaginative components brought in by psychosis, could not be ascertained.

The appearance or disappearance of hallucinations under the influence of factors that modify cerebral blood flow suggests that hallucinations in schizophrenia should not be regarded as entirely psychogenic in origin, and that metabolic changes, by making one cortical area or another abnormally active, can be responsible for their occurrence. This concept should not be regarded as negating the rôle of emotionally charged incidents or thoughts in influencing the content of hallucinations.

The observations made here on the production of hallucinations or of sharp, brief visual reproductions of memories concomitant with decreased cerebral blood flow are of interest in relation to the occurrence of such psychologic phenomena in clinical conditions in which cerebral flow is diminished, such as circulatory collapse, or the chill phase of febrile reactions.[15]

SUMMARY

Inhalation of carbon dioxide may result in temporary disappearance of hallucinations in some patients with schizophrenia. Marked diminution in cerebral blood flow may result in reappearance of hallucinations in some schizophrenic patients in whom hallucinations were present in the past; when cerebral blood flow is decreased in subjects who never had hallucinations, the subjects may experience brief, vivid visual images of events that happened in the distant past. The possible significance of changes in cerebral blood flow in regard to the appearance or disappearance of hallucinations in various conditions is indicated.

RESUMEN

La inhalación de anhídrido carbónico puede dar lugar a que desaparezcan en forma temporal las alucinaciones de ciertos enfermos de esquizofrenia. La marcada disminución del riego sanguíneo cerebral puede hacer que reaparezcan las alucinaciones en ciertos pacientes que las habían tenido en el pasado; en personas que nunca tuvieron alucinaciones, se presentaron breves y vívidas imágenes visuales de sucesos ocurridos con bastante anterioridad, como consecuencia de la mencionada disminución del riego sanguíneo cerebral. Se indica el posible significado de los cambios ocurridos en la circulación cerebral con respecto a la aparición y desaparición de las alucinaciones en diversas circunstancias.

RESUME

L'inhalation d'anhydride carbonique peut se traduire par la disparition temporaire des hallucinations chez certains sujets souffrant de schizophrénie. La diminution marquée de la circulation sanguine cérébrale peut entraîner la réapparition des hallucinations chez certains sujets schizophrènes qui en étaient affectés dans le passé. Lorsque la circulation sanguine cérébrale est ralentie chez des sujets qui n'ont jamais souffert d'hallucinations, il est possible que ces sujets aient des images visuelles très nettes, mais brèves, d'évènements survenus dans le passé lointain. L'auteur indique les conséquences possibles des modifications de la circulation sanguine cérébrale par rapport à l'apparition ou à la disparition des hallucinations dans divers cas.

ZUSAMMENFASSUNG

In manchen schizophrenischen Patienten bewirkt das Inhalieren von Kohlensäure ein zeitweiliges Verschwinden von Halluzinationen. Dagegen kann eine betonte Reduzierung der zerebralen Blutzufuhr in anderen Patienten, die einmal Halluzinationen hatten, diese wieder hervorrufen. Wenn die Blutzufuhr zum Gehirn in Versuchspersonen, die nie an Halluzinationen litten, verkleinert wird, können vorübergehende lebhafte Visionen von Ereignissen, die lange in der Vergangenheit zurückliegen, hervorgerufen werden. Die etwaige Bedeutung von Anderungen in der Blutzufuhr zum Gehirn in Bezug auf das Hervorrufen und Verschwinden von Halluzinationen wird angedeutet.

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Comparison of Decamethonium Bromide (C10) and D-Tubocurarine as Preventives of Traumatic Complications in Electroshock Therapy

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Since the successful introduction ten years ago into clinical medicine of curare as a preventive of traumatic complications in electroshock,[1] many drugs have been offered as substitutes. Beta erythroidine, magnesium sulfate, quinine methochloride, myanesin and seconal have all been found ineffective or harmful in side effects.

Recent investigators have advocated decamethonium bromide or C10, a synthetic muscle relaxant. Hobson and Prescott[2] found no difference between d-tubocurarine and C10 in degree of relaxation or sparing effect upon respiration, but favored C10 because of its short lasting effects and lack of histamine-like side reactions. In pre-electroshock treatment of 13 patients, Grob, Holaday and Harvey[3] listed shorter duration of action, more rapid recovery of motor power, less pronounced effects on pharyngeal, laryngeal, facial and in some cases respiratory muscles and no histamine-like side effects as advantages of C10 compared with d-tubocurarine. Unna,[4] in physiological experiments in humans, showed a greater depression of vital capacity by C10 than by d-tubocurarine at comparable dosage.

British opinion on the use of the two drugs in convulsion therapy and in abdominal surgery is divided. Davies and Lewis[5] gave C10 with thiopentone to 18 patients before electroshock, without disagreeable side effects. However, Gray[6] reports discontinuing the routine use of C10 as a muscle relaxant in abdominal surgery because of delayed respiration exchange in 4 cases and the smaller margin between adequate relaxation and apnea, as compared with the use of d-tubocurarine. A *Lancet* editorial,[7] pointing out the absence of a satisfactory antidote, the possible sharp fall in blood pressure and occasional delayed recovery, concluded: "It is not yet clear that C10 possesses any advantage over the natural drug."

New reports by Holt and coworkers[8] and by Moench[9] conclude that C10 is the drug of choice for pre-electroshock relaxation, though both investigators warn of danger in its use by the inexperienced and stress the necessity for skill in using intratracheal intubation with the anoxic patient. Further, Moench was unable to confirm the selective sparing of respiratory muscles reported by others in the use of C10.

A recent editorial[17] emphasizes the need to make comparisons of the clinical usefulness of such drugs as d-tubocurarine and decamethonium only under conditions of actual use, since otherwise serious misleading results may be obtained.

In view of these controversial opinions we have compared the use of C10 with d-tubocurarine in a larger number of patients than previously reported and by methods carefully evaluated for

C10 was supplied by Burroughs Wellcome and Company; d-tubocurarine by Endo Products, Inc.

objective, accurate collection of data. Our standard for comparison is the technic developed during ten years' satisfactory use of d-tubocurarine in all patients receiving convulsive shock.

To evaluate the new agent, C10, we used quantitative methods insofar as possible, based on our preliminary observations of its action as compared with d-tubocurarine in normal subjects. Control groups of patients were arranged, with the use of dynamometer and sphygmomanometer readings to measure the degree of relaxation, concealment from one of the two investigators of the drug used, and exact measurements of time intervals. Notes include subjective observations of pretreatment distress, amounts of salivation and strength of clonic contractions. Because of these added precautions,[10] descriptions of procedures are necessarily more detailed than usual.

METHODS OF PROCEDURE

1. Preliminary investigation on normal subjects attempted to establish the dosage and minimal satisfactory period for injection for C10 and the intervals until optimum relaxation was obtained. The aim was to adjust the dosage of both drugs in such a way as to attain an equal degree of relaxation. An injection-time of one minute was satisfactory for both drugs, and an interval of three minutes from the end of the injection to the time of treatment was found necessary for the maximum effect to be reached at optimal dosage.

Long experience has shown the optimum level to be one which will entirely eliminate traumatic complications, without causing the patient undue pretreatment distress or respiratory embarrassment. Descriptively, the patient can raise the arm but not the head; grip strength as measured by dynamometer decreases 85-95 per cent under the pretreatment performance; the ensuing convulsion is so modified as to eliminate outcry, opisthotonus, or raising the legs from the bed; and throughout the clonic phase the forearms can be easily extended by the observer when held only between the thumb and one or two fingers.

Recommended dosages of C10 vary greatly, from 0.75 mg. to 3 mg. (Moench)[9] to 4-5 mg. (Holt et al).[8] Effects of different dosages have not been reported, except for the statement that the effect of C10 does not vary with the patient's weight, as does that of d-tubocurarine; and that variability in response is greater than that to d-tubocurarine, according to Unna.[4]

We therefore had to devise our own method of establishing optimum dosage. We gave a minimum dose of 1.2 mg. (the minimum amount found to overcurarize slightly our most sensitive patient), measured by dynamometer the response at one-minute intervals and gave additional amounts at two-minute intervals until adequate grip depression was reached. This clumsy method, often requiring up to eight minutes, was the only one found to establish a dose without risking dangerous overcurarization or an essentially unmodified convulsion. For example, a dose of 1.4 mg. of C10 overcurarized a young, vigorous female, whose optimum dosage proved to be 1.2 mg.; whereas 3.4 mg. gave barely adequate relaxation to another patient.

The dosage of d-tubocurarine was based on the well-established principle of body weight, modified slightly for the degree of muscular development, nutrition, sex, age and especially the degree of agitation and overactivity. Myasthenia gravis is known to be a contraindication.

In order to minimize variables, no sedative or other medication was used during the preconvulsive period.

2. A Smedley dynamometer (Stoelting) measured grip strength as an index of diminished strength in the voluntary musculature. A Tycos sphygmomanometer, with hook-type blood pressure cuff (numbered and lettered for exact duplication of position), applied around the right biceps with the forearm at a right angle, and inflated to a constant pressure of 80 mm., indicated the strength of the clonic contraction after electrical stimulation.

3. Of the two observers (L. G. McK. and R. E. T.) one gave the injection and made objective measurements while the other, from whom the given drug was concealed, estimated subjective findings.

4. The drugs C10 and d-tubocurarine were given to each patient in the same number of doses, arranged either alternately or in blocks of four treatments each. That is, a patient received four treatments with C10 and four with d-tubocurarine, or the drugs were alternated in the treatment course.

5. To obtain a significant number of cases, all patients received both drugs over a five-month period. The first treatment with each drug was used to estimate the dosage and familiarize the patient with the procedure, and was excluded from the analyzed data.

6. Subjective observations included estimated pretreatment distress due to throat symptoms, strength of clonic contractions and estimated amounts of ensuing salivation. These signs were graded according to increasing severity in five steps, from 0 to 4 plus. At first both observers separately estimated the most important sign, the strength of contraction, in 59 treatments, in 55 of which the estimations were identical. In the other four the difference was one step. The coefficient of correlation (Pearson) between the two sets of observations was .93. Thereafter, only the uninformed observer made the estimations.

7. Objective tests made before, during and after treatment were as follows: One hour after breakfast the patient, put to bed, was asked to squeeze the dynamometer twice with his right hand with maximum effort; the higher of the two values was recorded as his initial effort. After the minute-long injection of the previously established curarizing dose, the second investigator, who did not know the drug used, during the next three minutes estimated the severity of the throat symptoms and the time at which the patient could no longer raise his head. Meanwhile the first physician, after one, two and three minutes, recorded dynamometer readings to measure the relaxation of the uniformly fatigued muscle group of the right forearm. After two and a half minutes a single reading was recorded from the left, unfatigued hand, to compare the effect of the two drugs on fatigued and unfatigued muscles. Just prior to electrical stimulation the hook-type blood pressure cuff was applied to the right arm at the previously established tension, the forearm was flexed at a right angle and the cuff was inflated to 80 mm. pressure. While the second observer, with a Rahm machine, gave the minimal stimulation for a *grand mal* convulsion and estimated the strength of clonic phase, the first observer recorded the maximum sphygmomanometer reading during the tonic contraction. These two observations made possible a comparison of the effects of the two drugs upon both the clonic and tonic phases of the convulsion. That is, a difference in the effect upon fatigued and unfatigued muscles should appear clinically as a difference in the tonic and clonic phases. The interval between the induction of the current and the first successful respiratory effort was measured by stop watch. No mechanical or physical restraint was used throughout the series.

Late in the study the question of concealment was tested. In 78 of 115 instances the supposedly ignorant observer correctly guessed the drug used. Analysis showed the highly significant chi square value of 14.6; only 6.635 is needed to show significance at one per cent. Signs that helped the observer to guess correctly were a different pattern of convulsion, gradually decreasing in severity with d-tubocurarine and increasing with C10; and a sign, first observed by a nurse assistant, that at the end of the convulsion with C10 the patient's eyes sometimes opened wide and pupils were dilated. These differences were slight, were noted gradually as the study continued and did not make differentiation specific.

Only data collected from these exact procedures were included in the comparative study.

The purpose for recording both left and righthand grip is important. Preliminary observation of C10 indicated a possible difference in action which might affect its relaxant qualities. It is now known that d-tubocurarine parallels myasthenia gravis in action;[11] further, d-tubocurarine and fatigue have similar effects upon voluntary muscles.[12] In our clinical experience we have observed that myasthenia gravis, fatigue and d-tubocurarine are synergistic or at least additive in their action.[13] Patients who receive C10 do not follow closely the syndrome of myasthenia gravis, and since the effect of C10 upon fatigue and upon myasthenia gravis is not clear, we felt that quantitative measurements of curarization should compare muscles after a constant amount of exercise with unfatigued muscles. We therefore compared the effects of C10 and d-tubocurarine on the depression of grip strength in five maximal efforts of the right hand during the test and in a single maximal effort of the left hand after curarization.

For statistical purposes the recorded grip strengths were expressed as percentages of depression from the maximum grip. For example, if a patient's maximum grip was 25 kg. and his weakened grip was 5 kg. the depression would be 20/25 or 80 per cent, recorded as GD 80.

Totals in number of patients and treatments: By this methodology the effects of C10 and d-tubocurarine were compared in 680 treatments of 95 patients during a five-month period. These private patients were treated for acute psychoses in the psychiatric department of a general hospital. Diagnoses were mainly depression, with some manic and schizophrenic patients. Most of the patients were cooperative.

Since the goal was a comparison of C10 and d-tubocurarine in the same patient at the same level of relaxation, treatment pairs were matched according to equal strength of clonic contraction. In case of multiple choice the rule was to match those treatments closest in time, or if equidistant, to reject the later one, thus avoiding prejudiced selection. A large number of single treatments at different levels of relaxation could not be thus matched. In 34 patients 122 treatments were found to match in 61 pairs, of which 38 were at optimal relaxation (2+), 15 at excessive relaxation (1+) and 8 at insufficient relaxation (3+). The matched comparisons concern these 122 treatments.

RESULTS

Similarities in action: Since such side effects as pretreatment anxiety, the degree of posttreatment salivation and the effect of respiration concern the desirability of relaxant agents, these factors were estimated. In our series only slight differences appeared in pretreatment anxiety, which patients expressed principally as throat distress, and in posttreatment salivation. These

side effects varied greatly from patient to patient; in the same patient they usually varied directly with the degree of relaxation. A comparison of matched pairs at each of the three levels of relaxation showed slightly greater throat distress with d-tubocurarine and slightly greater salivation with C10 (see Table 1). In this series we avoided the use of other drugs, as stated earlier. However, it is well known that small doses of pentothal in conjunction with either d-tubocurarine or C10 eliminate excessive pretreatment anxiety, while atropine reduces excessive posttreatment salivation.

Much conflicting opinion has disputed the effects of the two drugs upon respiration. We measured the duration of postconvulsion apnea as an index of respiratory effects, but did not attempt to measure respiratory volume. For both d-tubocurarine and C10 the postconvulsive apnea, measured from the time of electrical stimulation to the time of the first respiration, was the same (see Table 2). With proper dosage of both drugs and a clear airway, the strength of posttreatment respiratory efforts was adequate. Only with an overdosage of either drug should pharmacologic antidotes, oxygen administration or artificial respiration be necessary, though these facilities should always be at hand.

TABLE 1

Pretreatment Anxiety and Posttreatment Salivation

(Mean of estimations on 0 to 4+ scale at three levels of relaxation in 61 matched pairs of treatments.)

Strength of Contraction	1+	2+	3+
Pretreatment anxiety			
D-tubocurarine	1.7	1.3	.7
C10	1.2	1.2	.6
Posttreatment salivation			
D-tubocurarine	1.3	.9	1.1
C10	1.5	1.0	1.4

TABLE 2

Postconvulsive Apnea

(Comparison of mean apnea in seconds at three levels of relaxation, total of 242 treatments.)

Strength of Convulsion	1+	2+	3+
D-tubocurarine	54.8	49.5	49.6
C10	54.4	49.4	51.2

With both drugs paresis is adequate to prevent traumatic complications, without dangerous respiratory or circulatory paralysis, though any variation from the optimum dosage can carry considerable hazard. Neither drug affects the convulsive threshold—a decided advantage over barbiturates.

Difference in action: Our results showed that C10 relaxed an unfatigued muscle to about the same degree as a fatigued muscle, in marked contrast to the effect of d-tubocurarine, in which the unfatigued muscle showed considerably less relaxation than did the fatigued muscle (see Table 3).

Clinically this difference is important in the consistently greater strength of the tonic phase, as shown by higher sphygmomanometer readings with d-tubocurarine as compared with those under C10. The fact that most fractures occur during the tonic phase makes this greater relaxation obtained by C10 during the first contraction important (see Table 4). A difference in the

pattern of the convulsion, appearing especially under minimal dosage of both drugs, was that with d-tubocurarine the convulsion gradually diminished in strength, whereas with C10 muscular contraction increased during the convulsion.

Any comparative study of curarizing drugs must allow for repeated measurements in experimental situations, or the resulting unstandardized degrees of fatigue will invalidate the compared standards of relaxation.

TABLE 3

Effect of Fatigue

(Comparison of effects on partially fatigued muscles (GD) at 1, 2, and 3 minutes and unfatigued muscles (LGD) at 2½ minutes after injection. Mean percentage of grip depression at three levels of relaxation.)

	Fatigued			— Unfatigued	= Effect of fatigue
	GD 1	GD 2	GD 3	LGD 2½	$\frac{GD\ 2 - GD\ 3}{2} - LGD$
At convulsion of 1+ strength, 15 matched pairs					
D-tubocurarine	58	88	95	78	13
C10	52	81	93	88	−1
At convulsion of 2+ strength, 38 matched pairs					
D-tubocurarine	50	78	87	60	22
C10	34	61	74	72	−5
At convulsion of 3+ strength, 8 matched pairs					
D-tubocurarine	37	76	85	33	47
C10	42	61	68	54	10

TABLE 4

Strength of Tonic Contraction

(At three levels of clonic contraction, in 61 matched pairs of treatments, mean rise of pressure in sphygmomanometer cuff from 80 mm.)

Strength of Clonic Contraction	1+	2+	3+
D-tubocurarine	109	137	181
C10	63	74	112

An important known difference is the fact that d-tubocurarine has a pharmacologic antagonist, while at present C10 is without an effective antidote. With d-tubocurarine, neostigmine (0.5 to 1 mg.), given intravenously, has sufficient specific antagonistic effect so that even rather large overdosages can be corrected. Although early reports indicated that pentamethonium iodide (C5) antagonizes C10, Unna[4] has shown its failure to diminish the effectiveness of C10 in unanesthetized man. He also confirmed the well known antagonistic effect of neostigmine on d-tubocurarine.

Most writers, in comparing the two drugs, emphasize the histamine-like effects seen with d-tubocurarine. Many writers have picked up this fallacious observation from early reports. With optimum dosage and the modern purified preparation of d-tubocurarine we have seen this reaction neither in the present series nor in the past ten years of routine use, involving thousands of treatments. Actually, we saw the effect clinically in our very early experience with imperfectly purified curare infusions. These carried small amounts of resins to which we ascribe the histamine-like reaction. Modern chemical assay of d-tubocurarine minimizes variations in strength of the product and thus eliminates the need for a synthetic.[14]

In our use of C10 we found, as have others, that the patient recovers from the curarizing effects more rapidly than with d-tubocurarine. In our use of conventional electroshock therapy this rapidity is a definite disadvantage in that many patients, regaining full strength before they have recovered from posttreatment confusion, require restraint by nurses and attendants. With d-tubocurarine the pharmacologic restraint usually lasts as long as does the treatment confusion, thereby reducing overactivity and avoiding the need for physical restraint.

Unsatisfactory dosage: A principal difficulty with the use of C10 was the establishment and maintenance of satisfactory dosage. Despite our complicated method of trying to fix individual dosage, since the effect cannot be estimated in advance, we found over or undercurarization to an undesirable or even dangerous degree more than twice as often with C10 as with d-tubocurarine. In 340 treatments with each drug, patients were twice dangerously and 10 times moderately overcurarized with C10, as compared to four instances of moderate overcurarization with d-tubocurarine. It is noteworthy that both instances of severe overcurarization with C10 occurred with minimal doses of 1.4 mg. and 1.8 mg. In undercurarization four patients had essentially unmodified convulsions under C10 while two were insufficiently relaxed under d-tubocurarine.

In a few cases we found fairly large and unpredictable variations in the effect of C10 on the same patient. This occurred both with and without intervening treatments of d-tubocurarine, whose antagonistic effects are thereby ruled out. For example, one patient was adequately relaxed and had an optimum (2+) convulsion with 2.1 mg. of C10; about 10 days later a dangerously strong convulsion without apparent relaxation after a dose of 3 mg.; the next week adequate curarization again, with 3.4 mg. Such an increase in tolerance we found in about 10 per cent of the cases. In another patient a dose of 2 mg. of C10 on four successive days resulted in a (1+) convulsion and a 97 per cent GD on the first and third days; a (3+) convulsion and only 31 per cent GD the second; and a (2+) convulsion and 88 per cent GD the fourth day. Sphygmomanometric readings showed the strength of the tonic phases as successively 50, 80, 50 and 70 mm. elevation, consistent with the other readings. In our series the dosage of C10 ranged from 1.2 to 3.4 mg.

In contrast to this unpredictable variation with C10 in some patients, a uniform effect of d-tubocurarine is attained by considering the degree of the patient's activity and agitation, which increases the effect of a given dose, while weight gain decreases the effect.

An additional factor is uniformity in the time necessary to reach maximal relaxation. With insufficient dosage of C10 the peak of relaxation had consistently passed and recovery begun at three minutes after the end of injection period, whereas adequate dosage did not produce peak relaxation before three minutes. The smaller the dose, the earlier the maximum relaxation. Since inadequate dosage of C10 is not at once discernible, one risks giving electric shock therapy not only in the presence of too small a dose but after the peak effect of that dose. The ensuing convulsion, essentially unmodified, may easily result in traumatic complications. In contrast, the peak effect of d-tubocurarine is never reached before three minutes. This more uniform rate of maximum relaxation and slower recovery makes timing of electric shock therapy with d-tubocurarine less difficult. The longer maximum action is a disadvantage only in case of overcurarization of such severe degree as to require the antidote, neostigmine.

One unsatisfactory reaction had to do with respiration. In the total series of 680 treatments

of 95 patients, four middle-aged or older men had an unusual degree of cyanosis with insufficient respiratory exchange, marked slowing of the heart rate and occasional stridor. Others have attributed this type of reaction to extreme vagal stimulation by the electrical current.[15] It has been observed by one of the writers (L. G. McK.) in previous experience with uncurarized electric shock therapy. The reaction did not correlate with the degree of relaxation. In two patients it appeared with d-tubocurarine and not with C10 curarization; in the other two it appeared with equal severity with each drug. The significance of this reaction is not clear.

A minor note is the observation that both drugs were used with satisfactory results in a poliomyelitis patient who had paresis of all muscles of the trunk and extremities; both drugs were also used with no variation of usual dosage in several patients during insulin coma.

DISCUSSION AND CONCLUSIONS

Our study and the methods by which we safeguarded the approaches to observation clearly show certain differences in action that have not been noted or emphasized elsewhere. If these factors are not taken into account the results of the comparison are vitiated.

First, we have shown that fatigue does not affect the relaxation produced by C10 while it does affect the relaxation produced by d-tubocurarine. This means that any method relying on serial performances of a single muscle group as indices of progressive relaxation will not give valid measures of the degree of relaxation in unfatigued muscles in comparing d-tubocurarine with a drug of such dissimilar action as C10.

We found another aspect of this difference in action in the greater relaxation obtained during the tonic phase of convulsion with C10, at any particular level of grip depression, and the corresponding degree of severity of clonic contraction, since the effect of C10 is maximal with the first contraction and does not permit one strong muscle contraction before reaching maximal effect, as does d-tubocurarine. This *fact* establishes in *theory* that protection from traumatic complications can actually be obtained with C10 with what appears to be a somewhat stronger clonic contraction than with d-tubocurarine. Our difficulty in establishing dosage with C10 at present makes this advantage of theoretical importance only.

A second difference is in the uniformity of response to a given dose. Effective dosage of C10 varies greatly and unpredictably from patient to patient. This variation was found to be as great as 300 per cent. Since the difference between under and overcurarization in an individual patient is obtained by a change of dosage of less than 20 per cent, it is obvious that this great variation in action makes satisfactory dosage very difficult.

Even in the same individual the effect of a given dose of C10 has been seen, in some cases, to vary considerably and unpredictably from day to day. This variation sometimes exceeds the span of optimal effectiveness and results in unpredictably severe and surprisingly soft convulsions. From long experience and numerous experimental studies the factors affecting dosage of d-tubocurarine are known to be body weight, degree of muscularity, age, sex, fatigue, agitation and myasthenic reactions. Consequently the effect of the dosage of d-tubocurarine can be accurately predicted by one experienced in its use.

It has been repeatedly shown that C10 has a much shorter duration of action than does

d-tubocurarine, and this has been generally regarded as an advantage for C10. However, we found that the extremely transient action of minimal doses of C10 makes it difficult to give the treatment at the peak of relaxation, and in conventional electric shock therapy may necessitate extra restraint of the patient because he regains normal strength while still in a state of post-shock confusion.

We saw no sparing action on respiratory muscles with C10 as reported by some writers.

It is important, in using any drug to modify a procedure such as electric shock therapy, that the dangers of the drug be fewer than the dangers of unmodified treatment. We have contended for many years that d-tubocurarine, when properly used, is safe. In this series we met no reactions with either drug that seemed to endanger life, though twice, with small doses of C10, extreme relaxation was produced to a degree that made artificial respiration by oxygen insufflation necessary. We doubt that these two patients would have survived the large doses recommended by some writers. Although in our series use of C10 was safe, we believe this safety was due to careful individualization of the dosage.

At least one death has been reported. In the case of a man aged 42, undergoing an operation for the removal of part of a kidney, allergy to the drug C10, coupled with slight operative shock, is given as the cause of death. The patient received a total of 11 mg. in 3 doses.[16]

Thus the safety of C10 is not entirely established at present. The difficulty in establishing optimum dosage and the lack of pharmacologic antagonists indicate a smaller margin of safety than with d-tubocurarine, unless other advantages are present. In a few cases of middle-aged or older patients it was the opinion of one of us (R. E. T.) that these patients recovered from electric shock therapy with less cyanosis under C10 than under d-tubocurarine. Except for these isolated observations, we saw no evidence in our series of advantages that would offset those of d-tubocurarine.

The search for a more satisfactory substitute for curare in shock therapy has been, in our opinion, fruitless. This search arises from misconceptions about the drug or inexperience with its action.

D-tubocurarine fulfills all the criteria as a safe, satisfactory muscular relaxant and is tailor-made for this purpose. Other drugs, including C10, can be used for the same purpose, but have no significant advantages, and up to the present some definite disadvantages, as we have shown.

SUMMARY

The value of any new relaxant for prevention of traumatic complications in electroshock can be adequately assessed only by carefully controlled comparison with d-tubocurarine in a sufficiently large series. The method must take into account differences in pharmacologic action. Such comparisons of the new drug C10 with d-tubocurarine in 680 treatments show the following:

1. Paresis was as adequate with C10 as with d-tubocurarine.
2. No simple method of determining optimum dosage was found for C10. It is established for d-tubocurarine.

3. C10 has an unpredictably variable effect in some patients with repeated dosage.
4. At optimum dosage the effects on circulation and respiration are similar, and neither drug increases the convulsive threshold.
5. Differences in side effects of pretreatment anxiety and excessive salivation were so slight as to be unimportant. Histamine-like reactions were not seen with either drug.
6. The shorter action of C10 was no advantage in our use of conventional electric shock therapy.
7. C10 has at present no adequate pharmacologic antagonist.
8. Fatigue does not change the action of C10, while it increases the effect of d-tubocurarine.
9. Both drugs are relatively safe if competently used. Safeguards are well established with d-tubocurarine, but are less so with C10.

D-tubocurarine therefore still remains the drug of choice, compared with C10, for the prevention of traumatic complications in electric shock therapy.

RESUMEN

El valor de todo nuevo relajante en la prevención de las complicaciones traumáticas del "shock" eléctrico puede ser sólo evaluado por medio de una cuidadosa comparación con la d-tubocurarina en grupos de enfermos suficientemente numerosos. El método debe tener en cuenta las diferencias que existen en cuanto a la acción farmacológica. Sobre las bases mencionadas, la comparación hecha entre la nueva droga C10 y la d-tubocurarina, en 680 casos, muestra lo siguiente:

1. La paresia fué tan adecuada con C10 como con d-tubocurarina.
2. No se ha encontrado un método simple para determinar la dosis óptima del C10. Dicho método existe para la d-tubocurarina.
3. En algunos pacientes en quienes se repite la dosis, el C10 posee un efecto variable, que no se puede predecir.
4. A la dosis óptima, los efectos circulatorios y respiratorios son similares; ninguna de ambas drogas eleva el umbral convulsivo.
5. Las diferencias en lo que atañe a ciertos efectos secundarios, tales como la ansiedad previa al tratamiento y la excesiva salivación, fueron tan pequeñas que se las juzga como carentes de importancia. Con ninguna de las drogas se observaron reacciones "histamínicas."
6. La acción más corta del C10 no significó una ventaja en nuestro empleo del "shock" eléctrico convencional.
7. No existe, por el momento, un antagonista farmacológico adecuado para el C10.
8. Ambas drogas son relativamente inocuas si se las usa con habilidad. La protección contra los efectos indeseables de la d-tubocurarina se encuentra bien establecida, no así en lo que respecta al C10.
9. La fatiga no modifica la acción del C10, en tanto que aumenta la de la d-tubocurarina.

Por lo tanto, la d-tubocurarina sigue siendo la droga de elección—si se la compara con el C10—para la profilaxis de las complicaciones traumáticas de la terapéutica por el "shock" eléctrico.

RESUME

La valeur de tout nouvel agent de détente pour prévenir les complications traumatiques provenant d'électro-choc ne peut être estimée que par une étude comparative soigneusement contrôlée par rapport à la d-tubocurarine portant sur un groupe suffisamment grand. La méthode utilisée doit tenir compte des différences en action pharmacologique. Des comparaisons de ce genre entre la nouvelle drogue C10 et la d-tubocurarine dans 680 traitements ont donné les résultats suivants:

1. La parésie est aussi adéquate avec la drogue C10 qu'avec la d-tubocurarine.
2. Aucune méthode simple de détermination de la dose optimum n'a pu être trouvée pour la drogue C10. Elle est établie pour la d-tubocurarine.
3. La drogue C10 a un effet variable imprévisible chez certains sujets sous l'action de doses répétées.
4. Pour la dose optimum, les effets sur la circulation et la respiration sont semblables, et ni l'une ni l'autre de ces drogues n'augmente le seuil convulsif.
5. Les différences en ce qui concerne les effets secondaires d'anxiété de pré-traitement et d'excès de salivation sont si faibles qu'on peut les considérer négligeables. Ni l'une ni l'autre de ces drogues n'ont donné lieu à des réactions du genre de l'histamine.
6. La réaction plus courte de la drogue C10 n'a pas présenté d'avantage dans notre emploi de la thérapie classique par choc électrique.
7. La drogue C10 n'a actuellement aucun antagoniste pharmacologique adéquat.
8. L'une et l'autre des drogues sont relativement sans danger si on les emploie correctement. Les moyens de protection sont bien établis pour la d-tubocurarine, mais moins bien pour la drogue C10.
9. La fatigue ne change pas l'action de la drogue C10, alors qu'elle augmente l'effet de la d-tubocurarine.

La D-tubocurarine reste donc la drogue à préférer, par rapport à la drogue C10, pour la prévention des complications traumatiques en thérapie par choc électrique.

ZUSAMMENFASSUNG

Im Hinblick auf bestehende Meinungsverschiedenheiten über den Wert des künstlichen Muskelerschlaffungsmittels Dekamethoniumbromid (C10) als Ersatz für Kurare, verglichen die Autoren die Wirksamkeit von C10 und D-Tubokurarin bei der Verhinderung traumatischer Komplikationen in der elektrischen Schocktherapie. Unter Einhaltung von vorsichtig bewerteten quantitativen Methoden wurde der Vergleich an weitaus mehr als den bisher berichteten Fällen angestellt. Innerhalb von 5 Monaten wurden insgesamt 680 Behandlungen an 95 Patienten einschliesslich von Kontrollgruppen beobachtet. Jeder Patient erhielt dieselbe Anzahl von Dosierungen beider Mittel, entweder abwechselnd, oder in Serien von je 4 Spritzen. Die erste Behandlung, die lediglich zur Bestimmung der Dosis diente, wurde jedesmal von den zu analysierenden Resultaten ausgeschlossen. Es wurde versucht, den gleichen Erschlaffungsgrad mit beiden Mitteln zu erreichen. Alsdann wurde eine Methode entwickelt, mit der die optimale Dosis von C10 gemessen werden konnte. Griffstärke wurde in ermüdeten und unermüdeten

Muskeln vor, während und nach der Behandlung durch ein Dynamometer gemessen. Um einen Vergleich zwischen ermüdeten und unermüdeten Muskeln zu gewährleisten, wurde die Griffstärke der rechten und linken Hand gemessen. Ein vorher geeichtes Sphygmomanometer diente dazu, die Stärke der klonischen Kontraktionen nach der elektrischen Reizung festzustellen. Subjektive Bewertungen befassten sich mit dem aus Rachensymptomen vor der Behandlung erwachsenden Unbefinden, mit der Stärke der klonischen Kontraktionen und mit dem Grad der erfolgenden Speichelabsonderung. Zuerst machten beide Beobachter diese Schätzungen unabhängig von einander; später wurden sie lediglich von dem Beobachter gemacht, der nicht wusste, welches Mittel angewandt worden war.

Die Resultate zeigten, dass beide Mittel eine genügende Erschlaffung erzielten. Optimale Dosierungen hatten in beiden Fällen ähnlichen Einfluss auf den Blutumlauf und die Atmung, und keines der beiden Mittel erhöhte die Krampfschwelle. Unterschiede in den Nebeneffekten auf die Angstlichkeit des Patienten vor der Behandlung und auf übermässige Speichelabsonderung waren unwesentlich. Keinerlei histaminähnliche Reaktionen wurden beobachtet. Deratige Reaktionen traten zwar in Erscheinung, als D-Tubokurarin zuerst in Gebrauch kam, sollten jedoch mit der heutigen Verfeinerung dieses Mittels wegfallen. In geübten Händen erwiesen sich beide Mittel als ungefährlich.

C10 hat den Nachteil, dass die Resultate bei verschiedenen Patienten und manchmal bei demselben Patienten im Verlauf der Behandlung schwanken und nicht genau vorauszusehen zu sind. Während die Methode der optimalen Dosisbestimmung bei D-Tubokurarin feststeht, ergab sich keine derartig einfache Methode für C10; und ausserdem gibt es gegenwärtig kein zufriedenstellendes pharmakologisches Gegenmittel für C10, wie das mit D-Tubokurarin der Fall ist. Dies ist ein wichtiger Punkt, da eine Ermüdung den Effekt von C10 unverändert lässt, während sie den von D-Tubokurarin verstärkt. Ferner ist der Einfluss des C10 auf Krankheiten wie Myasthenia gravis unbekannt. D-Tubokurarin bleibt daher im Vergleich mit C10 das Mittel der Wahl zur Verhinderung von traumatischen Komplikationen in der elektrischen Schocktherapie.

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Diagnosis, Psychodynamics, and Treatment — A Point of View

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Experience suggests that there are wide differences in the understanding of the meaning and scope of the terms, diagnosis and psychodynamics. For the purpose of this communication, primary interest does not rest on the diagnostic labels that would place psychiatric cases into appropriate diagnostic classifications. It is understandable that when confronted with a combination of, let us say, depressive and obsessive reactions, psychiatrists will aim to reach a uniform diagnosis. To do so, they tend of necessity to focus their attention more specifically either on the depressive or on the obsessive reaction. Yet from the patient's point of view both elements are of significance. Psychodynamically too their temporal relationship may be striking as well as significant—as in cases in which depressions occurring after obsessions are subsequently alleviated with the clearing of the obsessions. Irrespective of the relationship of the reactions, the obvious fact remains that both are prominent in disturbing the patient. One may, therefore, be closer to a comprehension and factual formulation of the patient's troubles if one describes his condition as a combination of depression and obsession reactions.

Such a formulation, leading to a hybrid diagnosis—depression and psychoneurosis—may be troublesome for one who is expected to subordinate his diagnostic evaluation to the requirements for the classification of mental disorders. But therapeutically it would certainly be more beneficial to the patient. For in this era of specialization, when shock treatment is regarded by some as the specific treatment for depression—assuming for the moment that they are correct—it is of the utmost importance to recognize significant psychoneurotic conditions.

Psychodynamics is concerned with the mental processes underlying personality functioning: One speaks of *psychic determinism*, implying that certain personality reactions and symptoms of mental disorders are outward manifestations of mental processes of which the individual is either completely unaware, or only more or less aware. That the unconscious plays an important part in the formation of certain symptoms of mental diseases is fairly generally admitted. For some, however, the unconscious remains the all-powerful force responsible for both certain everyday life reactions of the average normal individual and certain abnormal reactions in mental disorders.

My own feeling is that the overemphasis on the rôle of the unconscious often results in unwarranted neglect to recognize fully the potency of the *reasoning person versus the feeling person in the eternal struggle between the pleasure principle and the reality principle.*

Another contention which, to my mind, also suffers when the significance of a principle that is basically sound is exaggerated, is that experiences in early childhood are essentially, if not exclusively, the determining factors for the adult personality function. It is generally recognized that early childhood is singularly receptive to influences from the immediate environment, for at

least two obvious reasons: the entire dependence of the child upon that environment for the gratification of his biological needs; and his inability to meet environmental influences critically, because of his lack of previous experience.

It seems to me that overemphasis on the psychodynamic effects of early childhood experiences detracts from a recognition of the importance of other equally valid factors. My understanding is that environmental influences begin as early as the intrauterine life; that they effect the progeny very significantly in early childhood, but remain effective throughout the life of the individual; that in all periods of life there is consistently interaction between personality and environment—circular reactions.

Let us consider a patient who has been subjected for a number of years to many painful experiences in her married life. Is it not possible that their cumulative effects may be dynamic features just as potent as the assumed, but not elicited, early childhood experiences? Thus the meaning of psychodynamics is broadened, in that it includes the mental processes caused, not only by early childhood experiences, but equally by experiences throughout the pre-illness life of the patient. To be more specific, I should like to add that life experiences at *any* age may be valid psychodynamic factors *per se*, regardless of whether or not they may be reasonably connected with certain childhood experiences. Such an understanding of psychodynamics contributes to both diagnostic evaluation of the patient's troubles and their treatment.

The guiding principle of rational or genetic-dynamic psychotherapy is the recognition that the patient's present-day reactions are somehow connected with his past life experiences. In accordance with the meaning of psychodynamics as here conceived, the psychotherapist will consider as significant material for psychotherapeutic discussions any and all emotional traumas which have occurred, not only in early childhood, but at any time throughout the life of the individual. His method of treatment therefore, will be the analysis of the patient's personality function in the present and throughout his past life.

In these general remarks there is no room for a discussion of what the content of the psychotherapeutic discussions should be; it is self-evident that the content will be as varied and polymorphous as human experiences can be, i.e., that it will take the form of ideas, fleeting or more consistent thoughts, or day and night dreams. I do want to stress the point that this concept of the meaning of genetic-dynamic psychotherapy—the counterpart of etiologic-pathogenetic therapy in medicine—leads to a recognition of the dynamic significance, not alone of the emotional traumas, but equally of two other factors—one within and the other without the patient—which can and should be used for therapeutic purposes:

The factor within the patient is habit formation: Because of past emotional upheavals, conscious or unconscious, the patient develops modes of behavior which either exclude certain of his usual daily activities, or include newly acquired unusual reactions. The patient's abnormal behavior is thus maintained by both the original genetic-dynamic factors—traumatic experiences, and the resulting modes of behavior which gradually became habits integrated into the personality function. The genetic-dynamic treatment should, therefore, be paralleled by active encouragement of the patient to begin to practice gradual dehabituation, from the time that he is, in the psychotherapist's judgment, sufficiently prepared for it. It seems to me that if the therapist waits for

the completion or near completion of the psychotherapeutic discussions, expecting that the patient's difficulties will then vanish at once, *globally*, he will be giving way to unwarranted passivity. This allows the patient to maintain his troubles without impressing upon him the necessity to use actively the insight he has gained in trying to provoke some break in his abnormal behavior—even though, to begin with, a very modest break.

The other factor lies in the patient's present environment: its positive or negative psychotherapeutic influence cannot be too strongly emphasized. Granted that the patient himself may be wholly, or more or less significantly, the maker of his environment, the fact remains that a given environment, *per se*, may be helpful or harmful to a given individual. To ignore the part which the present-day environment plays in the patient's difficulties, in the belief that when his internal conflicts will be resolved by the psychotherapeutic sessions, he will be able to adjust to the environmental adversities, means failure to recognize a significant etiologic factor. Not to be concerned about the environment is, therefore, to deprive psychotherapy of additional support; and in certain cases even to allow the bad environment to weaken, if not to neutralize altogether, its accomplishments.

RESUMEN

El clínico trata de llegar a un diagnóstico uniforme en las enfermedades cuyos síntomas son característicos de trastornos psicopatológicos muy diferentes. En los casos parecidos sería mejor formular un diagnóstico "híbrido" debido a que una descripción exacta de los trastornos que padece el enfermo puede sugerir un tratamiento apropiado.

En esta communicación, *psicodinámico* significa *procesos mentales* inducidos no sólo por los acontecimientos que han afectado al paciente desde su infancia sino también por las pruebas sufridas en el transcurso de su vida entera. Los "choques" emotivos que afectan a un individuo no importa a qué edad, pueden convertirse en factores psicodinámicos muy importantes, aunque se considere que ellos no se encuentran en manera alguna relacionados con trastornos emotivos de su infancia.

Por lo tanto, no se encuentra ciertamente justificado el considerar al *subconciente* como de importancia primordial, relegando la *conciencia* a un lugar secundario, subordinada a la *subconciencia.*

Nuestra concepción psicodinámica nos lleva, así, pensar que el principio más importante de la psicoterapia racional es el análisis de las funciones del enfermo sus perturbaciones emotivas presentes y pasadas.

Sin embargo, además de los traumas emotivos, otros factores desempeñan un papel dinámico y en consecuencia deben ser considerados desde el punto de vista terapéutico.

Uno de ellos es el desarrollo de un modo de conducirse y de reaccionar anormalmente a consecuencia de "choques" emotivos sufridos por el enfermo antes de su enfermedad actual. Por lo tanto, junto con las sesiones psicoterapéuticas, el enfermo debe ser inducido a tratar de liberarse en forma gradual de sus hábitos enfermizos, tan pronto como sea capaz de hacerlo.

RESUME

Le clinicien tâche d'arriver à un diagnostic uniforme même dans les cas où't les symptômes sont caractéristiques de conditions psychopathologiques fort différentes. Dans des pareils cas

un diagnostic hybride est indiqué de fait qu'une description exacte des troubles dont le malade souffre peut suggérer un traitement approprié.

Dans cette communication, *psychodynamique* signifie *mental processes* induits non seulement par des évènements qui ont affecté le malade dans son enfance, mais également par des épreuves personnelles durant sa vie entière. Des chocs émotifs frappant l'individu à n'importe quel âge peuvent devenir des facteurs psychodynamiques très importants, même si l'on considère qu'ils ne sont d'aucune façon associés avec des troubles émotifs d'enfance.

Par conséquent, il n'est certainement pas justifiable de considérer la *subconscience* d'être d'importance primordiale, reléguant ainsi la *conscience* à une place secondaire, subordonnée à la subconscience.

Notre conception psychodynamique nous amène donc à penser que le principe fondamental de la psychothérapie rationnelle, c'est l'analyse de la fonction du malade, ses perturbations émotives, dans sa vie actuelle et passée.

Cependant, en plus des trauma émotifs, deux autres facteurs jouent un rôle dynamique et par conséquent doivent être considérés au point de vue thérapeutique:

L'un est le développement d'une façon de se conduire et de réagir anormalement à la suite des chocs émotifs subis par le malade antérieurement à sa maladie actuelle. Par conséquent, parallèlement avec les discussions psychothérapeutiques, le malade est encouragé de tâcher de se libérer graduellement de ses habitudes maladives, dès le moment où il est capable de le faire.

ZUSAMMENFASSUNG

Der Kliniker bemüht sich darum, auch in solchen Fällen einheitliche Diagnosen zu erzielen, in denen gewisse Reaktionen intensiv und häufig genug sind, um verschiedene psychopathologische Zustände zu charakterisieren. In solchen Fällen ist eine Zwitterdiagnose angebracht, weil aus der wahrheitsgetreuen Formulierung der Schwierigkeiten des Patienten die Behandlung folgt.

Die Bedeutung der Psychodynamik wird dadurch erweitert, dass man als gleichwertige dynamische Faktoren nicht nur Erfahrungen aus frühester Kindheit ansieht, sondern auch Erfahrungen aus dem gesamten Vorleben des Patienten. Lebenserfahrungen können an sich machtvolle psychodynamische Faktoren sein, gleichgültig in welchem Alter sie wurzeln und ob sie im Zusammenhang mit Kindheitserfahrungen betrachtet werden oder nicht. Eine zu grosse Betonung des Unbewussten erscheint daher unzulässig, besonders wenn sie, wie das häufig geschieht, zu einer Unterschätzung des Bewussten führt.

Dem hier umrissenen Begriff der Psychodynamik nach wäre die Analyse der Funktion der Persönlichkeit des Patienten, in der Vergangenheit und Gegenwart, die Hauptrichtlinie einer rationalen oder genetisch-dynamischen Psychotherapie. Es handelt sich jedoch nicht nur um das gefühlsmässige Trauma, dem dynamische Bedeutung beigemessen und das in der Therapeutik ausgenützt werden soll, sondern auch um zwei weitere Faktoren: die Bildung von Gewohnheiten und die gegenwärtige Umgebung des Patienten.

Es wird zugegeben, dass in dem anormalen Verhalten des Patienten sowohl die ursprünglichen traumatischen Erlebnisse als auch die daraus fliessenden Reaktionsweisen, die allmählich zu mit der Persönlichkeit verschmolzenen Gewohnheiten werden, eine Rolle spielen. Der Patient wird daher ermutigt, allmähliche Entwöhnung zu üben, sobald er dafür genügend vorbereitet ist.

Graphic Art as a Special Ability in Children With a Reading Disability

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Reading disability represents a clear-cut defect in the visual motor field. Children with this disorder are capable of recognizing all forms except written language. Sometimes they have difficulty recognizing certain letters, while often they read single letters well but are not able to read words consisting of several letters. In severe cases even three letter words like "cat" and "dog" present barriers which the children cannot overcome. Often difficulties arise through the confusion of similar letters such as "b" and "d" and "p" and "q." Children with this difficulty sometimes start to read the word in the opposite direction, as though they were reading it in mirror image, which may explain why "b" is mistaken for "d" and "p" for "q." Some investigators, such as Samuel T. Orton,[8, 9] see in this reversal tendency the essential basis for the reading difficulty. It is interesting that, oftentimes, numbers can be handled more easily than words or letters. In addition such children always show a special incapacity to relate the visual structure of a word to its auditory structure. This is probably due to the difficulty of experiencing the total auditory and visual gestalt of the word as a sign.

In children with a specific reading disability or congenital alexia the other optical functions are intact. In a study by Paul Schilder,[10] pictures were exposed by a tachistoscope for a very short time (1/100 to 1/10 second) to children with reading disabilities, and it was shown that their optic perception was in no way inferior to that of the normal reader. It was emphasized, therefore, that this is an isolated disorder in gnostic-intellectual functioning in which the written word and letter cannot be integrated and differentiated, especially in relation to the spoken word; and there is also difficulty in differentiating the spoken word into its sounds or in putting the sounds of the word together. There are often special difficulties in differentiating the letter from its mirror image.

Immaturity in the patterning of optic perception and also in postural or motor behavior are revealed in studies by A. A. Silver.[11, 12] There is often a defect in right-left discrimination and in the finger schema. Perceptually the visual motor gestalt function is relatively primitive.[1, 2] There seems to be some primary difficulty in differentiating the foreground from the background. The body image concept is often impaired or immature as shown by the human figure drawings (Goodenough). The immature integration of tonus and posture, the right-left-handedness conflict, and difficulties in spatial orientation are also shown by projection in the human figure drawing.

The tendency for horizontally directed configuration to take the vertical position or "verti-

*This paper was written before Paul Schilder's death (1940). Some additions have been made at this time from the more recent contributions in the literature to the subject under discussion.

calization" has been found by A. A. Fabian[5] to be compelling as an immature fixation in reading disabilities.

The motor immaturity of reading disabilities seems to be comparable to that of stuttering as shown by Helene Kopp.[7] However, there is no actual inability to carry on complicated motor behavior. The capacity of such children to do handicrafts is not impaired, and indeed they often showed a special interest in fields of visual motor activity other than reading, and only this particular world of signs is slow in maturing. It is largely a difficulty in the sign function, since words can be correctly copied. The structure of the word, the sign, is less accessible to them. The printed or written sign, which is so important to us all, does not mean a specific sound; it is not a specific object for them to which they can have a specific attitude. In this way, such children are excluded from that part of the world from which most of us receive important experiences. It is not surprising that they turn with great eagerness to the part of the visual motor world which is open to them. Indeed, we find some such compensatory reactions in a great number of cases. They take a particular pleasure in a colorful world of fantasy, invent fantastic stories, do elaborate handicraft work, and some even become accountants, transferring to numbers some of the pleasure others have in words. If school children with reading disabilities have some such outlet and are not continually pushed to school tasks for which they are not fitted, they may present no special problem. But if these outlets are not open to them and they are not given remedial training in reading, or if the reading disability is taken as a sign of intellectual inferiority or stubbornness (see Abram Blau[4]), the child starts a justifiable defensive struggle and becomes a behavior problem.

There have been many studies on the personality maladjustments of children with reading disabilities. Phyllis Blanchard has approached the problem psychoanalytically and believes that the reading disability is not a primary, but a secondary or neurotic symptom.[3] It is suggested that the reading content, which the child turns away from through the disability, is associated either directly or symbolically with unconscious emotional conflict.

The reading disability has been related by Robert E. Stewart[13] to personality problems, such as poor motivation due to an insecure relationship with parents or as an expression of hostility against an ambivalent parent. In Rorschach studies by Edith Gann[6] more pure color responses, color shock and dark shock and poor form visualization were found.[8] These Rorschach signs were interpreted as indicating immaturity and a disturbance in emotional development and social adaptation.

Children with reading disabilities certainly have personality problems and neurotic reactions. There appear, however, to be few common factors except a relative immaturity and a need to build up some defense mechanisms or compensations. Furthermore, a simple understanding does not help such a child sufficiently in his fundamental difficulty, and psychotherapy by itself is not enough. In most cases the reading disability is a developmental retardation, and with more or less specific aid the child eventually learns to read. Sometimes this development does not come in time, and the education of the child may be severely hampered. Even in severe cases, remedial teaching is helpful to an orientation in the structure of the written word. In mild cases the difficulty may disappear, although it is probable that the attitude of the child toward the world will remain different from that of children who are not afflicted with this developmental handi-

cap. In such children psychotherapeutic help may be beneficial even with specific tutoring. The difficulty appears to be due to a lag in development of the specific functional patterning in the parietal and occipital areas of the brain. Children of high as well as low intelligence may be equally affected. The following case is a well-defined example of this kind.

Marcellus was a boy of nearly average intelligence. His behavior difficulties started in school when he was forced into an occupation of which he was not capable, namely, reading. He immediately sought an outlet in scribbling. The particular gifts which he displayed in his pictures are probably in close relation to the fact that he concentrated the energy on drawing which normally would have been used for other purposes.

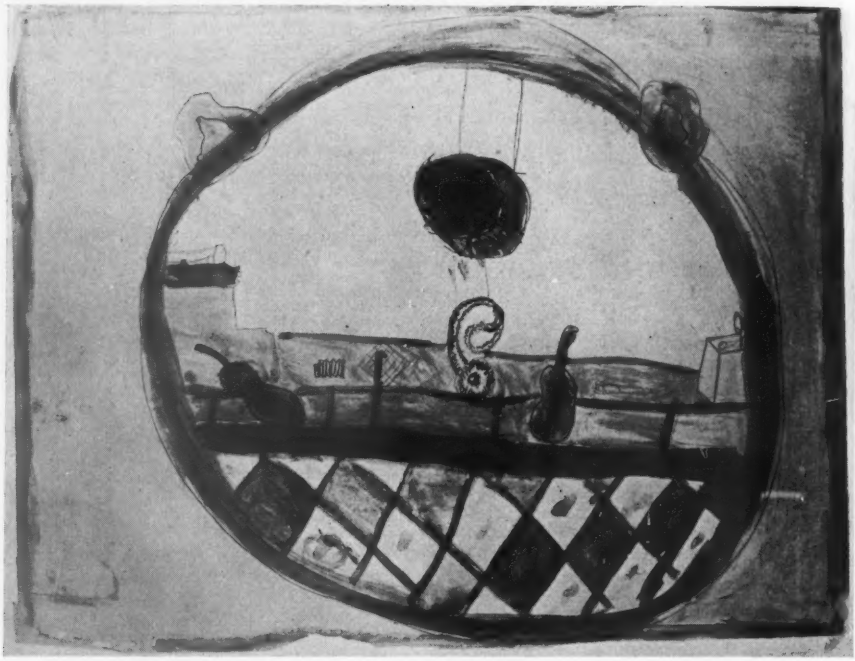
He was an 11-year-old boy who was referred to us by his school with the complaint that he was unmanageable. The report stated that he would not attend to any of his lessons; when given books, he would not study them but destroyed them with scribbling. He became sullen and defiant to any correction by his teachers and was unfriendly and aggressive toward other children. His mother said he had been a good and happy child until he started school, but that since then he had become progressively more defiant and disobedient to her and quarrelsome with a younger sister.

Our study revealed that the was slightly duller than the average child. He had adequate intelligence to do fourth-grade work, could do third-grade arithmetic, but could not do even first-grade reading and spelling. He was left-handed, but his master eye was the right one, a rather common finding among children with a reading disability. We also found him a restless, unhappy child with marked feelings of inferiority. He said he hated school and played truant as often as he dared. He felt that he was dumb, and that his mother preferred his sister because she was brighter and did not cause as much trouble as he did. He fought with the other children because he felt they picked on him. He admitted he often scribbled on school books and other property in order to occupy himself. When placed in our art class, the true nature of his scribbling soon became apparent.

We will start with an analysis of this boy's chef-d'oeuvre—an orchestra score (Fig. 1). A great love for detail is noticeable, particularly in the careful drawing of the two violins and the trumpet. The approach is a realistic one, and there is a decided will-to-form in the picture. The margin of the paper is outlined in blue, while the picture itself is framed in a circle, which is quite accurately drawn. The composition of the instruments on the board admirably distributes the values in space. There is a framework of criss-cross lattices which support the board. The colors are subdued and well poised in relation to each other, with soft green and greenish blue predominating. Color is not independent and rampant, but rather a part of the total composition drawn by an individual who is unusually aware of space and measures it. In the first sketch of this picture the colors were a little deeper, but still formed a graceful symphony of colors. In the four corners of this smaller sketch there were four decorative designs in red.

The boy is at his best in drawings in which a colored margin and a circle give him a definite background. There is a real drive to put his drawings in a more or less perfect circle. However, he is not completely bound by a circular pattern. In the picture (Fig. 2) of a lion-tamer, in which he caught the attitude of the man and the posture of the lion in an excellent way, the figures are placed in a realistic landscape and executed in brown, green, and blue, with an almost

FIG. 1



classical distribution of masses. Among the many pictures of children we have studied, those of this boy are outstanding for the cleverness with which he distributed the space between background, foreground, and the individual parts of the study. They have an almost classical equilibrium. This applied equally to his crayon sketches and water colors.

He has an excellent feeling for human posture, and he draws, with the same skill, a man falling from a tree, a bearded priest, a lion-tamer, and a lady with a muff. It should be noted that the lion-tamer is wielding the whip with his left hand. In the crayon drawing of the priest praying for the people (Fig. 2) the lively colors fit the theme. The two upper corners contain decorative crosses. Here again we note the fine balance and proportion which his work displays. In another of the pictures which portrays an angel in yellow the decorative figures in the corners are more schematic and a little further removed from reality. It is interesting that even these more or less abstract figures, which are executed in brilliant and symmetrical colors, have something of a conventional form and order about them. This artist is certainly not a cubist or a surrealist, even though he occasionally uses abstract geometrical forms for decoration. Whereas most of the children were rather limited in the material they depicted, this boy covered a great part of the world in his work. There were boats, castles with turrets, landscapes, trees, bridges, lions, birds, angels, priests with all their paraphernalia, in addition to ordinary human beings.

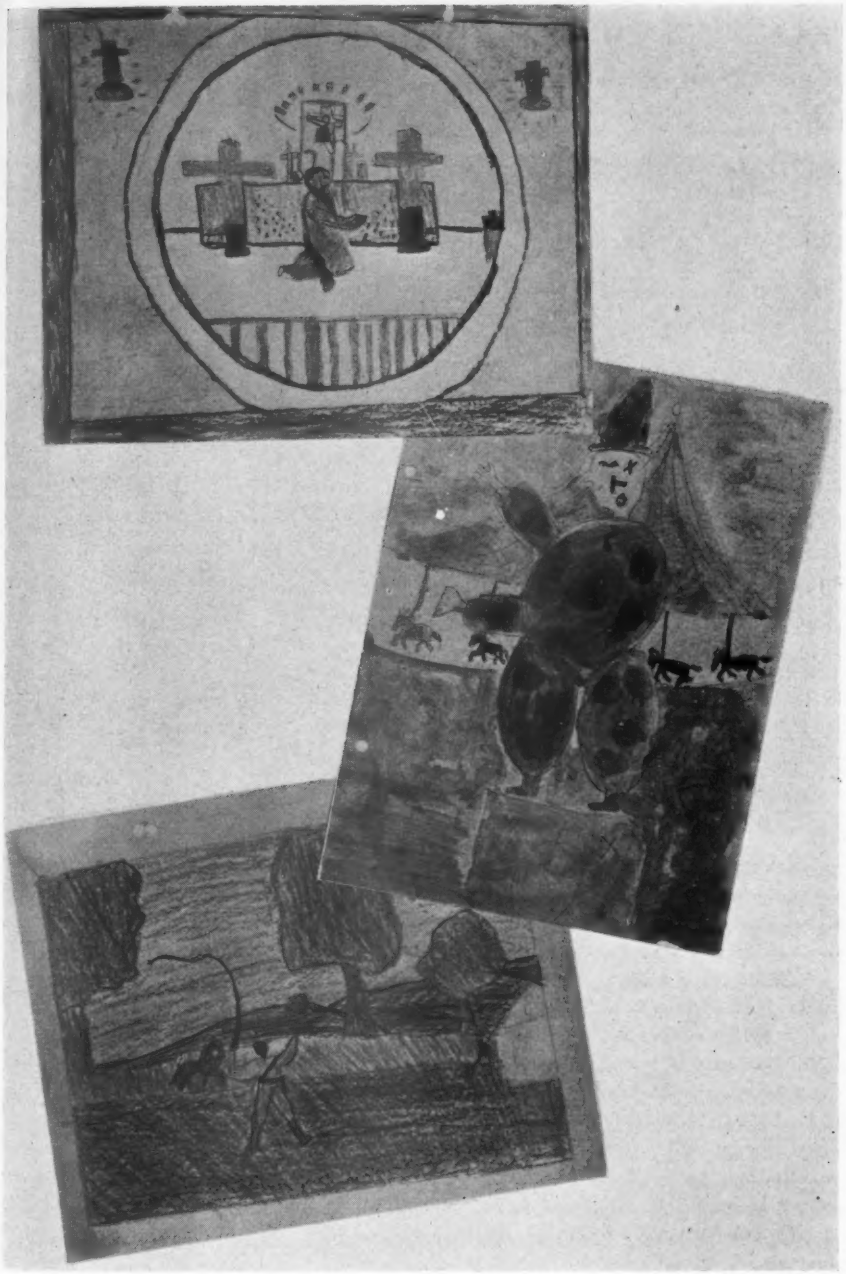
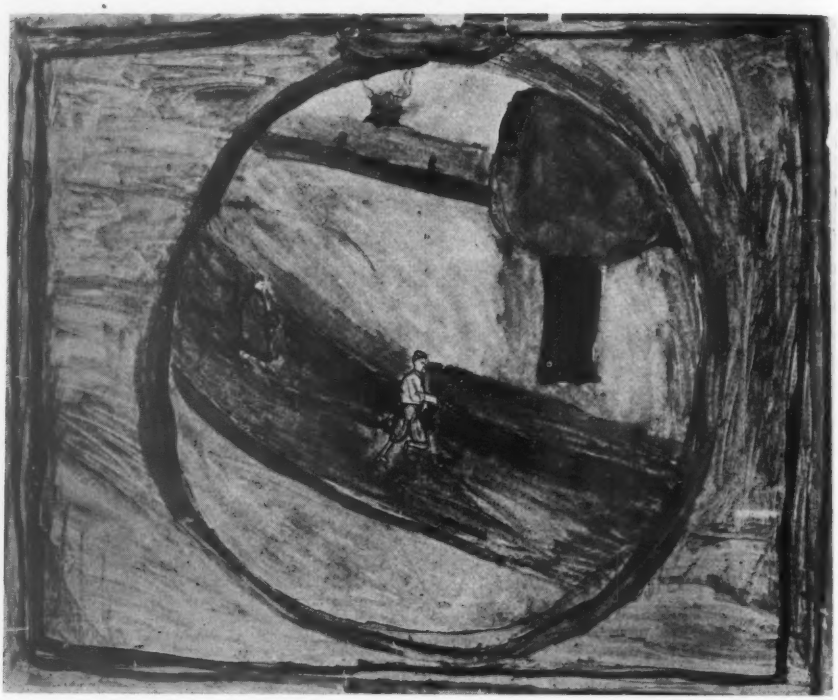


FIG. 2

FIG. 3



It is evident that the child desires reality and plenty of it. Color and form are equally important, and poise and equilibrium become his method to catch reality. The landscape shown in Figure 3 is a good instance of the principles discussed. It might be mentioned that, in addition to this boy's artistic talent, he also showed some dramatic ability.

A child with a reading disability, who is disappointed in the part of the reality which he cannot master through reading, is able to compensate by concentration on the world as he sees it and by other modes of expression which he can master. Among these methods of expression, those akin to writing are particularly dear to him. Such observation gives us a deeper insight into the fundamental psychology of those children who have reading disabilities.

In the case of Marcellus, his remedial training in reading soon convinced him that he could learn to read. We gave him the satisfaction and public recognition which his excellent graphic and dramatic art justified. When we returned him to his school, we advised that he be given proper materials and encouraged to "scribble" when his teachers were not able to give him lessons and instructions which he could grasp and use. We have had no further complaints from his school.

The scientist never likes to draw conclusions from one case. Fortunately, the material at

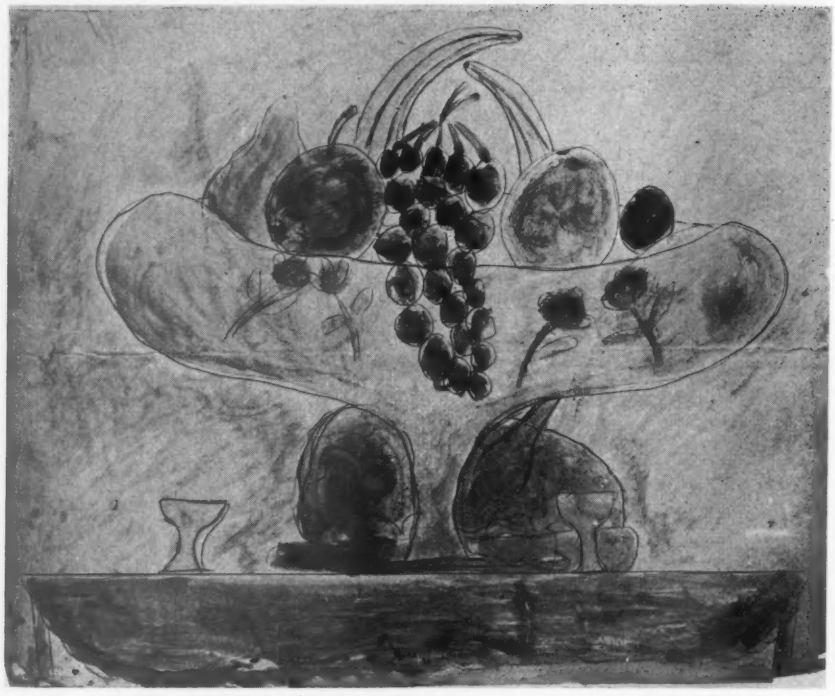
Bellevue Psychiatric Hospital is rich enough to offer several examples of children with problems similar to those of Marcellus. But we will discuss here only one other, a much younger child, Albert, whose untutored technical ability was as good as that of Marcellus. He was 8 years old but had the intellectual capacity of a 9½-year-old boy. He should have been able to do fourth-grade work, but was failing in the second grade because he could not read at all and his spelling was very poor.

This boy had had a very unsatisfactory life. His father had died when he was an infant, and his mother was an inadequate neurotic individual who complained that she could not take care of him herself, yet interfered when arrangements were made for boarding-home care. This had tended to foster an infantile attitude in an otherwise promising child. Furthermore, when he was 7 years old, his skull was fractured in a severe automobile accident. This became a focal point of anxiety for the mother and soon also for Albert himself. He believed it had injured his brain so that he could not learn and explained his reading difficulty on this basis, but his school records showed that the disability was evident before the injury. It did appear, however, that the head injury had lowered his margin of fatigability and rendered him more sensitive to unpleasant stimuli, thus encouraging his already existing infantile and hypochondriacal tendencies. In addition, one eye had been injured so that there was a slight blurring of vision which, needless to say, accentuated his difficulties. Such a combination of disabilities might seem almost insurmountable. Many of our case histories present evidence of such multiplicity of factors. Most children seem to be able to stand one handicap, but the accumulation of two or three may finally be more than the child can tolerate without responding by the development of a more or less serious behavior disorder. This boy would not attend school, he complained continually of headaches, dizziness, nausea, earache, and blurred vision. He would not play with other children, maintaining that he was afraid their rough play would injure him. He clung to his mother and "nearly drove her frantic," but she would not allow a boarding mother or school teacher to make any effort to push him into normal interests or activities.

The same problems faced us on the Children's Ward. He was unwilling to enter into any activities, expressed fear of the other children, and complained to his mother of mistreatment until she asked to remove him. Soon we found that he could do good work in the art class. This was a particularly fortunate ability for this boy, because it quickly showed him that even blurred vision due to the injury to his eye, a head injury which seemed to lower his working capacity, and a reading disability which prevented him from learning as other boys did could not be so serious after all.

There is one picture (Fig. 4) which he repeated over and over again, a basket filled with different fruit. Each drawing has its particular attractions in the different versions. The grapes which are realistically drawn were purple in the figure here outlined, but in another version they are brown. There is an equally delicate arrangement of colors in both drawings. The turkey at the bottom of the study does not especially add to the value of the work but was obviously dear to the child because of its special significance. Without it the picture is more successful from an artistic point of view. A third version shows tall candles on either side of the basket enhancing the color combinations which, with the reds, yellows, brown, green, and greenish yellow, are perfect.

FIG. 4



We can see that children with disabilities are capable of art expressions which are quite realistic even when they are concerned with such intricate form principles as Marcellus. It is almost like the work of Salvador Dali, where the fantastic content demands verification by realistic technic. This realistic technic by the children does not serve as reassurance that the fantastic exists, but gives the child confidence that he is capable of handling something of reality. The high level attained in this one picture was not always maintained by Albert. He also made a study of swans in a river, in which rather large and ungainly yellow and blue birds vie in ungainliness with green and red water lilies, and the figures in the foreground are out of proportion to the carelessly smeared trees in the background. However, the picture (Fig. 2) of the clown is delicate and masterful in the distribution between foreground and background. The artfulness in the drawing of the face reaffirms our belief in the artistic capacities of this boy.

When, in spite of a reading disability, a child can express himself in drawings which are as perfect as these, his self-confidence is increased, and this becomes an important aid in an otherwise difficult treatment. Such drawings should not be considered an avenue of escape but should

aid the child in his self-assurance that he may start anew in the task of reading in which he has been discouraged by the misunderstanding of the adult.

In the case of Albert, after a period of satisfying productivity in the art classes we were able to get him to attempt work in other activities and finally accept tutoring from a reading teacher, and he found that he was not as handicapped as he had come to believe. By means of good boarding home and school placement, which were possible because of the boy's unusual potentiality as shown by an intellectual test and his art work, he made a rather happy adjustment, only interrupted by one period of restlessness, which a little advice and reassurance from us cleared up rapidly.

RESUMEN

La incapacidad para la lectura en los niños de edad escolar se define como un retraso en el desarrollo y una inmadurez en las funciones gnóstico-intelectuales, en virtud de la cual la palabra escrita no puede integrarse y diferenciarse especialmente en relación con la palabra hablada. Existen también dificultades para diferenciar los sonidos de la palabra hablada, y con frecuencia hay otros signos de immadurez para establecer los cuadros de percepción visual motora, conducta postural y motora, y estructura de la personalidad. Sin embargo, la percepción visual con frecuencia no está retardada, mostrando muchos niños una capacidad para compensar su incapacidad específica con una habilidad especial en las artes gráficas. Se presentan las historias clínicas de dos de tales niños, junto con seis ilustraciones de sus trabajos de arte. Los autores estudian también el uso de esta capacidad artistica especial en el programa de entrenamiento educativo.

RESUME

L'incapacité de lecture chez les écoliers est défini, à la lumière des résultats d'autres travaux de recherches, comme un retard de développement ou d'immaturité du fonctionnement gnostic-intellectuel, de sorte que le mot écrit et la lettre ne peuvent pas être intégrés et distingués en particulier en relation avec le mot parlé. On observe souvent aussi des difficultés à distinguer les sons du mot parlé, et on constate fréquemment d'autres indications d'immaturité dans le degré de perception motrice visuelle, dans le comportement moteur et posture, et dans la structure de la personnalité. Toutefois, on rencontre souvent des cas où la perception visuelle n'est pas retardée, et nombreux sont les enfants qui manifestent une aptitude spéciale pour l'art graphique (dessin) compensant ainsi leur inaptitude particulière. Les observations faites pour deux de ces cas sont indiquées avec six exemples de leurs travaux de dessin. L'article traite aussi de l'utilisation de ce talent artistique spécial dans l'adoption d'un programme éducatif correctif.

ZUSAMMENFASSUNG

Auf Grund von anderen Forschungsergebnissen werden Störungen in der Lesefähigkeit von Schulkindern als verlangsamte Entwicklung oder Unreife der gnostisch-intellektuellen Funktion definiert, bei denen geschriebene Worte und Buchstaben nur unvollständig erfasst und unterschieden werden, besonders im Zusammenhang mit dem gesprochenen Wort. Solche Kinder haben auch oft Schwierigkeiten zwischen den Lauten der gesprochenen Worte zu differenzieren, und es zeigen sich Merkmale der Unreife in Bezug auf den Gesamtzusammenhang zwischen

visueller Wahrnehmung and motorischer Koordination. Die visuelle Wahrnehmungsfähigkeit an sich ist jedoch häufig überhaupt nicht gestört, und manche Kinder zeigen die Gabe, ihre spezifische Leseunfähigkeit durch ein besonderes Talent im Zeichnen und Malen wettzumachen. Die Fälle zweier solcher Kinder werden dargestellt, und sechs Beispiele ihrer Kunst werden reproduziert. Die Art, in der diese besondere künstlerische Fähigkeit in der Behebung von Lesestörungen ausgenützt werden kann, wird besprochen.

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Experimental Abridgment of the Rorschach Test

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PART I

The time-consuming nature of psychological projection tests is no doubt the main reason why they are used so little by psychiatrists; most psychiatrists, with very good reason, are unwilling to trouble to acquire a test technique that may well take more time than a psychiatric interview. Unless the test offers very substantial advantages it is obviously an uneconomic procedure, and from a practical point of view few psychiatrists would agree that projection tests are "worth their keep."

The usual compromise is that a psychologist does the actual testing and scoring, and passes the results to the clinician. This is not so revealing as obtaining the results at first hand, and so far as the time factor is concerned the burden is merely shifted to the psychologist. The ideal solution would be a rapidly-administered, easily-scored test, but the question always arises—can this be achieved without considerable sacrifice in precision and content?

In one type of projection test (Thematic Apperception) it has been pointed out by Valentine and Robin (1950) that certain stimulus-cards are more evocative for certain purposes than others; and it would therefore be worth while to evaluate test material with a view to excluding the less "valuable" items. How far this can be done in the case of the Rorschach test, it is the aim of this investigation to discover.

It was decided to test the feasibility of the hypothesis first by extracting abridged scores from a series of complete Rorschach test results. For this purpose the protocols in Beck's (1945) book were selected, as they are available to other investigators and also offer an exceptionally uniform type of scoring. The whole series of 47 protocols was used; it covers a wide intelligence range both in normal subjects and a variety of psychiatric syndromes.

The use of ten plates in the Rorschach test is based on custom and has an empirical foundation. As Rorschach himself pointed out, "the series of figures used in this test gradually developed on the basis of empirical observations. . . . While I do not mean to claim that this series is a 'non plus ultra,' I must emphasize that results, if they are to be comparable to those here presented, must be obtained with identical plates or with an analogous series suitably standardized." (Rorschach, 1942).

The difficulty of standardization has no doubt been one of the main difficulties in the trial of a shorter test. This question arose even in Zulliger's introduction of a control series of ten plates and they have never achieved much general popularity. In formulating a shorter test therefore, it seemed better to base it on the original Rorschach plates themselves.

Certain features would have to be kept in mind in so doing. The first plate, for instance, should preferably be a neutral gray-black with opportunity for white-space responses, so no change need be made from Plate I. To preserve as much similarity as possible with the original test, the next plate should be one of the black and red cards. This gives the subject an oppor-

tunity to show color shock, as well as to reveal movement responses. Either Plate II or III would suit here, so Plate II was retained as the second card in the *Abridged* series. In the ten-figure Rorschach test the series continues through four gray-black plates, IV-VII; of these, Plate VI was selected as the third plate of the *Abridged* series. The last three plates of the Rorschach test consist of multicolored figures, and for the first trial Plate X was selected. The *Abridged* test now consisted of Plates I, II, VI, and X.

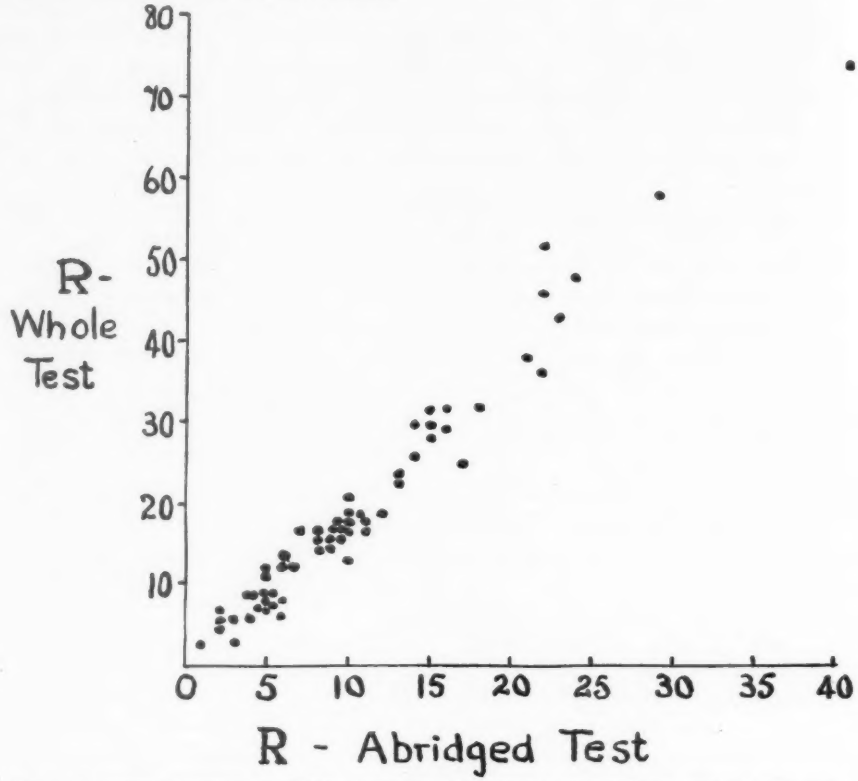

FIG. 1. A scattergram showing the number of responses (R) obtained in the *Whole* and *Abridged* scores over a series of 60 records. Coefficient of correlation = +0.98.

The scores for these four plates were accordingly abstracted from Beck's book, and compared with the scores for the *Whole* records (Plates I-X). Each scoring category was considered separately, and the method is demonstrated from the portion of a full table reproduced in Table 1; correlations were then worked out for some of the scoring categories and these results appear in Table 2. It will be seen that, although giving a satisfactory match in some respects, in others the degree of correlation is low. It was accordingly decided to add a fifth card to the short

TABLE 1

Abstraction of the scoring categories from the protocols.

	R		W		D		dd		s		
	Whole	Abridged	Whole	Abridged	Whole	Abridged	Whole	Abridged	Whole	Abridged	etc.
Record 1.	68	36	23	14	35	16	10	6	11	7	
Record 2.	58	32	8	4	39	22	11	6	6	4	
Record 3.	40	22	8	3	27	16	5	3	2	1	
Record 4.	91	43	5	2	43	19	42	22	10	3	
Record 5.	44	26	7	3	32	19	5	4	5	3	
Record 6.	29	17	4	2	19	11	6	4	1	1	
etc.											

series, Plate VIII, and the scores were abstracted again from the protocols. The *Abridged* series now consisted of Plates I, II, VI, VIII, and X. The means and standard deviations of the *Whole* and *Abridged* scores were recalculated, also the degree of linear correlation between them; these are shown in Table 3.

It will be observed that the correlations are fairly consistently good, and they will in fact stand comparison with the reliability of most psychological tests. The following extract from Vernon (1940) sets out the situation on the question of reliability correlations in the field of testing: "The typical correlation coefficient between results on ordinary two-hour papers in the same subject is known, from many experiments, to be no higher than +0.70. . . . In all these instances—namely repetition, application and scoring by a different tester, parallel form, and corrected split-half—we generally expect to get an intercorrelation of at least +0.90. For if this reliability coefficient is much lower it would indicate that the scores are too unstable to be trusted. Standardized educational and intelligence tests generally reach this level of reliability, but ordinary examinations often fail to do so."

Regression equations for the scoring categories of Table 3 are shown in Table 4. Regarding the forecasting of *Whole* scores from the *Abridged* test findings, these will be fairly accurate for groups, but the regression equation has always a considerable margin of error in the individual case. This margin of error can be conveniently estimated by the coefficient of forecasting efficiency, E, equal to $1-\sqrt{1-r^2}$ (see Garrett, 1947). Taking the case of the scoring category

TABLE 2

Coefficient of correlation (r) between Whole *record and* Abridged *record scores for a number of scoring categories.* (Abridged *Record consisting of Plates I, II, VI and X.*)

R	(Number of Responses)	+0.93
W	(% of Whole Responses)	+0.94
M	(Number of Human Movement Responses)	+0.49
Sum C	(Sum of Color Responses)	+0.83
F+%	(% of "Good Form" Responses)	+0.85
A%	(% of Animal Responses)	+0.83
Z	(Organization Score)	+0.78
P	(Number of Popular Responses)	+0.70
s	(Number of White-Space Responses)	+0.85

R (number of responses), forecasts of the *Whole* record score based on the *Abridged* score and the regression equation will be 80 per cent efficient. Forecasts based on the lowest correlation (for P, or Popular responses, where $r = +0.83$) will still be 44 per cent efficient in the individual case. In all types of individual-case forecasting based on regression, however, efficiency drops off rapidly as the coefficient of correlation falls, and r must be above 0.87 for the forecasting efficiency to be greater than 50 per cent.

TABLE 3

Showing the means and standard deviations for a number of scoring categories in the Whole *and* Abridged *records, and the degree of linear correlation between the* Whole *and* Abridged *scores. Total number of cases: 47. (*Abridged *test consisting of Plates I, II, VI, VIII and X).*

			Mean	Sigma	r
		Whole	35.94	15.91	
	R	Abridged	19.38	8.43	+0.98
		Whole	6.53	5.99	
	W	Abridged	3.30	3.13	+0.95
		Whole	24.40	7.19	
	D	Abridged	12.38	5.55	+0.98
		Whole	5.98	6.92	
	Dd	Abridged	3.66	4.00	+ .97
		Whole	67.51	14.84	
	F+%	Abridged	68.15	16.22	+0.90
		Whole	4.26	4.19	
	M	Abridged	2.11	2.38	+0.94
		Whole	3.82	3.28	
	Sum C	Abridged	2.49	2.24	+0.95
		Whole	13.56	7.55	
Raw Score	A	Abridged	8.04	4.42	+0.88
		Whole	39.66	15.41	
	A%	Abridged	41.83	17.02	+0.86
		Whole	5.36	2.66	
	P	Abridged	2.85	1.52	+0.83
		Whole	3.13	3.26	
	s	Abridged	1.66	1.94	+0.91
		Whole	34.84	30.42	
	Z	Abridged	18.99	16.79	+0.97

TABLE 4

Regression equations for the scoring categories listed in Table 3. A symbol followed by $_w$ *indicates the "Estimated Whole Score" which may be found by solving the equation with the* Abridged *Score,* $_a$*. The* $\pm$ *values are the standard deviations, not probable errors.*

R_w	=	1.85 R_a	+	0.09	$\pm$	3.18
W_w	=	1.81 W_a	+	0.56	$\pm$	1.92
D_w	=	1.27 D_a	+	8.67	$\pm$	1.44
Dd_w	=	1.68 Dd_a	+	2.43	$\pm$	1.73
$F\text{-}\%_w$	=	0.82 $F\text{-}\%_a$	+	11.63	$\pm$	6.53
M_w	=	1.76 M_a	+	0.55	$\pm$	1.49
Sum C_w	=	1.39 Sum C_a	+	1.33	$\pm$	1.05
$A\%_w$	=	0.77 $A\%_a$	+	7.45	$\pm$	7.86
P_w	=	1.45 P_a	+	1.23	$\pm$	1.49
s_w	=	1.53 s_a	+	0.59	$\pm$	1.34
Z_w	=	1.76 Z_a	+	1.42	$\pm$	7.60

TABLE 5
For explanation see text.

(1)	(2)	(3)
R	(W) — 44.3 (A) — 43.5	0.98
W	(W) — 91.7 (A) — 94.8	1.03
D	(W) — 29.5 (A) — 44.8	1.52
Dd	(W) — 115.7 (A) — 109.3	0.94
F+%	(W) — 22.0 (A) — 23.8	1.08
M	(W) — 98.4 (A) — 112.8	1.15
Sum C	(W) — 85.9 (A) — 90.0	1.05
A (raw score)	(W) — 55.7 (A) — 55.0	0.99
A%	(W) — 38.8 (A) — 40.7	1.05
P	(W) — 49.6 (A) — 53.3	1.07
s	(W) — 104.1 (A) — 116.9	1.12
Z	(W) — 87.3 (A) — 88.4	1.01

Another point of interest is whether, in reducing the number of plates, we have also increased the variability of the scoring. This can be checked by the coefficient of variation, which, expressed as $V = 100\ \sigma/\text{Mean}$, enables one to compare the variability of groups independently of the units in which they are expressed. In actual fact, it will be seen from Table 5 that there is a high degree of correspondence between the variability of the *Abridged* and *Whole* scores. Column (1) lists the scoring categories, column (2) shows the coefficient of the variation in the *Whole* and *Abridged* scores respectively for each category, and column (3) indicates the ratio variability of *Abridged/Whole* scores. For example, so far as the score category R is concerned, the *Abridged* records are 2 per cent less variable than the *Whole* records; this is a negligible error. As the mean variability ratio for the 12 scoring categories on Table 5 is 1.08 $\pm$0.14, it will be seen that for practical purposes the *Abridged* scores may be taken to vary around their means almost exactly as do the *Whole* record scores.

However, the scoring categories considered above do not cover the whole range of Rorschach scoring; others remaining include scores for Color (C), Color-Form (CF) and Form-Color (FC); Gray-black (Y) and mixtures of Form and Gray-black (YF and FY according to how far Form dominates); also Vista responses, scored V, VF and FV, again according to the relative domination of Form; and Positional responses, Po. These determinants are scored relatively infrequently, and the coefficient of correlation is scarcely relevant owing to the numerous zero scores. To estimate the efficiency of the *Abridged* test in recording these responses, a table was prepared

TABLE 6

Frequency table for scoring categories C. CF, FC. Y, YF and FY, VF and FV. (P. 01)

No. of times scored in Abridged Record	No. of times the Category was scored in the Whole Record										
	1	2	3	4	5	6	7	8	9	10	Total
0	25	8	1	—	—	—	—	—	—	—	34
1	31	26	6	5	—	—	—	—	—	—	68
2		15	9	4	—	—	1	1	—	—	30
3			3	2	1	1	—	—	—	—	7
4				2	2	1	—	—	—	—	5
5					2	1	—	—	—	1	4
6						—	—	1	—	—	1
Total	56	49	19	13	5	3	1	2	—	1	149

showing how often the category was scored in the *Abridged* test compared with the frequency of scoring in the *Whole* test. (See Table 6). It will be seen that when the category has a zero score in the *Abridged* record, the probability is that the *Whole* record score is 1; also that a zero score in the *Abridged* record was not found to accompany a *Whole* score higher than 2, except in one instance. Similarly an *Abridged* score of 1 is most likely to correspond with a *Whole* score of 1 or 2, and was not found accompanying a *Whole* score of more than 4. Also an *Abridged* score of 2 tends to correspond with a *Whole* score of 2 or 3, etc.

Going on to consider the scoring of each of these categories individually, Table 7 shows how "successful" the *Abridged* record is in recording responses scored in the course of the *Whole* test. Here we are considering not the frequency with which a type of response occurs, but the number of subjects who score it. If a "success" is counted when the category is recorded by the *Abridged* score, and a "failure" when it is not, the average of "successes" is about 79 per cent.

In considering the significance of these tables, it should be noted that the slight "inaccuracy" of the *Abridged* test may serve a useful purpose. That is, a score which is present to a moderate

TABLE 7

From the series of 47 cases, Row (1) indicates how many showed the scoring category in the Whole *record cards (I-X). Row (2) indicates how many of these showed the same scoring category in the* Abridged *record, and Row (3) expresses this as a percentage of the total. The mean percentage of scoring in the* Abridged *record is 78.6%; weighted mean 77.5%.*

	C	CF	FC	VF or FV	YF or FY	Y	Po
(1) Scored in Whole Record (No. of cases)	20	29	31	23	33	13	2
(2) Scored in Abridged Record (No. of cases)	16	23	26	21	23	6	2
(3) %	80	79	84	91	70	46	100

or marked degree in the *Whole record* is rarely missed by the *Abridged* test; but when a *Whole* test score is only feebly represented it tends to drop out altogether in the *Abridged* record. It is conceivable that in the longer test the extra score-scatter gives the subject more opportunity to conform with the normal response-pattern; so the tendency of the shorter test to exaggerate deficiencies may be a feature worthy of investigation.

Summary

This section may be summarized as follows: In an attempt to find whether significant inaccuracies would be produced in shortening the Rorschach test, the scores for four plates (I, II, VI and X) were abstracted from the 47 protocols published by Beck. Correlations between these *Abridged* scores, and the *Whole* scores of which they form a part, ranged from +0.93 to +0.49.

When another plate was added to the *Abridged* series (now Plates I, II, VI, VIII and X) a higher degree of correlation was found between the *Abridged* and *Whole* scores. The variability of scoring was not significantly altered, and the *Abridged* records failed to register only scores which were feebly represented in the *Whole* records.

PART II

The foregoing results give at least a reasonable basis of expectation that the Rorschach test may be shortened without the introduction of drastic inaccuracies. However, it must be remembered that the results were obtained by abstraction from *Whole* records obtained in the usual way, and it does not of necessity follow that in actual practice the same correlations will be found.

The first point that arises is this: is the *Abridged* test reliable in the sense of being consistent within itself, is its self-correlation satisfactory? Ordinarily this question can be answered by the employment of a variety of techniques: test-retest, parallel form, split-half, or "rational equivalence" (Garrett, 1947). These methods cannot be applied directly to the Rorschach, however. In the test-retest or repetition method, the findings after an interval of time are reasonably constant only in a stabilized normal, or possibly in a stabilized psychotic. The parallel-form method offers great difficulty in standardization, the split-half method is scarcely applicable owing to the difficulty of sectioning two comparable halves, and "rational equivalence" does not apply either, as none of the items are directly interchangeable. Lacking an adequate measure of the reliability of the full Rorschach test, we are unable to calculate by the Spearman-Brown "prophecy formula" how much lowering of reliability might be expected in halving the test. If we were to assume that the Rorschach reliability is 0.96, then halving the test would, by formula, reduce the reliability to 0.92.

However, the whole question of the reliability of the *Abridged* test is less important than its correlation with the full test. It would be of little interest to find that the *Abridged* test was more "reliable" than the full Rorschach if at the same time the validity showed a falling-away.

The method finally adopted to evaluate the practical efficacy of the *Abridged* test was by using it as part of the *Whole* test. In effect, this resolves itself into doing an ordinary Rorschach test with the plates in the following order—I, II, VI, VIII, X; IV, V, VII, III, IX. The scores of the first five plates (the *Abridged* test) may then be compared with the *Whole* test scores. The

objection may very properly be raised that this is not a normal Rorschach test procedure, as the plate order is changed. However, changing the sequence of the plates probably involves no more inaccuracy than would be met with in retest. Rabin and Sanderson (1947), for example, divided their subjects into two groups; in one, the test was given with the plates in their normal order (I-X), and in the other the order was reversed (X-I). After two months both groups were retested, each with the plates in the order opposite to the one they had had previously. No significant differences were found in productivity nor reaction-times by changing the order of the plates, and they state, "It seems safe to conjecture that the factor of retest is probably just as important in the variations (i.e. in any variations produced) as is the change in order of presentation." It will be noticed that the change in sequence introduced in the *Abridged* method is much less drastic than reversing the order of the series; in fact, every effort is made to preserve the usual full-test sequence of stimuli. It is all the less likely, therefore, that significant variations will be introduced by presenting the plates as has been done for the purpose of this study.

A standardized test procedure was followed, and of course the plates were always presented to the subject in the fore-mentioned order—I, II, VI, VIII, X; IV, V, VII, III, IX, with no break in continuity. When the protocols came to be scored, this was done in the usual way but after each score the *Abridged* score was added in parentheses; this consisted of course of the values for the first five plates only.

A total of 60 subjects in all were given the test in this way. The locale of the material varied; some cases were drawn from mental hospital practice—Runwell Hospital, Essex; Scalebor Park, Leeds; and Kingseat Hospital, Aberdeenshire. Other cases were from the medical wards of the Royal Infirmary, Aberdeen; and the bulk of the psychoneurotic records were obtained from the outpatient psychiatric clinic in Aberdeen. In passing, I would like to thank the clinical chiefs of these establishments for access to patients and the opportunity to test them. The breakdown of the total, from a diagnostic point of view, may be presented as follows:

Psychoneurosis	33
Depression	9
Schizophrenia	11
"Organic" disease	4
Subcultural amentia	3
	—
	60

The *Whole* and *Abridged* record scores were collected from these subjects and analyzed in a manner similar to that demonstrated in Part I of this paper. It should be noted in passing that the scoring system used in this section is not identical with Beck's, which was of course used in the first section where we were considering the protocols drawn from his book. I prefer to class animal movement responses under FM instead of including them under Form responses; also texture responses (Fc) and inanimate movement (Fm) have been used here although they are not identical with Beck's system. This makes no very essential difference; the only point is that Form response percentage in Part II is not directly comparable with F per cent in Part I. As we do not require direct comparisons between the sections, the matter is not relevant.

It may be suggested that some correlation is bound to occur between the whole and the part, in a test break-up of this description; so that no matter how the *Abridged* series is formed, some co-variance is to be expected. This is only true within limits. For a factor common to all plates, such as the number of responses (R), there will almost of necessity be some interdependence of *Whole* and *Abridged* scores; but a factor such as color can be scored only on certain of the plates. It would appear quite feasible, therefore, to construct an *Abridged* version which would correlate quite reasonably for R, poorly for F per cent, and not at all for CF and FC. Thus the automatic "whole-part" correlation lends nothing to the general balance of *Abridged*-test reliability; this can be accomplished only by selection of a suitable series of plates for the shorter version of the test. In point of fact, the mean correlation obtained is significantly higher than any expected whole-part correlation.

The *Whole*-test—*Abridged*-test correlations for a number of scoring categories are given in Table 8. It will be noted that all correlations are fairly high, and the mean is +0.93. The lower result for P, or Popular scores, is partly due to the small-range discrete scale. A sample scattergram is shown in Figure 1, to give a visual impression of one of the correlations.

TABLE 8

Coefficients of correlation between Whole *and* Abridged *tests, for certain scoring categories, over a series of 60 subjects.*

Scoring Category	Coefficient of Correlation
R	+0.98
W	+0.90
D	+0.97
F%	+0.90
F+%	+0.97
A%	+0.88
P	+0.78
Rt	+0.92

Mean $z = 1.669$, corresponding to mean $r = +0.93$

The less frequently scored responses do not lend themselves so readily to correlation treatment. Instead, a contingency table was prepared from which the amount of scatter may be observed (Table 9). The most probable *Whole* score for any *Abridged* score is underlined. The correspondence between the two forms of test for these categories is unfortunately rather less satisfactory than for the scores of Table 8. In the psychoneurotic group, for example, two subjects each scored two shading responses in the first five plates; one subject scored no further responses of this type whereas the other went on to register another three. The table therefore can provide only a rather approximate guide to these score-categories.

So far as the content of the responses was concerned, the position is more satisfactory. It was invariably found that any predominating type of content, displayed over the ten-card series, retained its majority in the *Abridged* test.

It is not suggested, on the basis of these results, that tables can be drawn up from which the "expected *Whole* score" can be derived from the "observed *Abridged* score." The inherent difficulty in such a procedure is that the correlation discrepancy may occur in critical instances. However, there seems to be little chance that a potentially abnormal, or very unusual, record,

TABLE 9

Contingency table showing the relationship between Whole *and* Abridged *scores for certain categories. The most common contingencies are italicized.*

Abridged Score	Whole Score 1	2	3	4	5	over 5
0	*FM. Fm. FK. Fc. C′ C. CF. FC M O + O − dr s*	FM. Fm. FK. Fc. C′ C. CF. FC. M O + O − dr s	FM. Fm. FK. Fc. C′ M s			
1	*FM. Fm. FK. Fc. C′ C. CF. FC* M O + O − dr s	FM. Fm. FK. Fc. C′ C. CF. FC *M* O + O − dr s	C. CF. FC M O + O − dr s	C. CF. FC M dr		
2		FM. Fm. FK. Fc. C′ *C. CF. FC* O + O − dr s	FM. Fm. FK. Fc. C′ *C. CF. FC* O + O − dr s	FM. Fm. FK. Fc. C′ C. CF. FC *M* O + O − dr s	FM. Fm. FK. Fc. C′ O + O − dr s	O + O − dr
3			O + O − s	FM. Fm. FK. Fc. C′ *C. CF. FC* M O + O − s	FM. Fm. FK. Fc. C′ C. CF. FC M s	M
4 or more						FM. Fm. FK. Fc. C′ *C. CF. FC* O + O − dr

will go altogether undetected within the space of the first five plates used in this way; in which case the remainder of the plates may be used to obtain fuller information.

In view of the general tendency to good correlation, interest attaches to those subjects who show discrepancies. As the *Abridged* portion constitutes to some extent a "test within the test," we can regard the subject's performance in this way:

Over the first 5 plates	Over the second 5 plates
i normal range	normal range
ii abnormal range	abnormal range
iii normal range	becoming more abnormal
iv abnormal range	becoming more normal

The commonly-occurring classes i and ii are the consistent scorers whose results correlate well; and those in the smaller classes iii and iv tend to produce greater or lesser correlation discrepancies. It remains to be seen whether any significant information can be gained from classing subjects after this fashion, and whether the type of scatter correlates with personality attributes. An investigation into this question is being pursued at the present time.

Summary

The Rorschach test was presented to a series of 60 subjects in the following order—Plates I, II, VI, VIII, X; IV, V, VII, III. IX. The first five plates constituted the *Abridged* test, which was correlated with the results for the *Whole* series. The mean correlation for the main scoring categories was +0.93; correlations for less commonly scored categories were less satisfactory; the main content was identical in the two aspects of the test. It is not considered that this form of abridgment can give an exactly reliable picture of the subject's expected performance over the complete ten plates, but something may be gained from the fact that within limits it constitutes a test within the test.

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RESUMEN

Parte I

Con el objeto de determinar si la simplificación de la prueba de Rorschach daria lugar a inexactitudes de importancia, se resumieron los resultados de 4 placas (I, II, VI y X) de los 47 protocolos publicados por Beck. La correlación entre estos resultados *resumidos* y los *completos*, de los cuales forman parte, varió entre + 0,93 y + 0,49. Se observó un mayor grado de correlación entre los resultados *resumidos* y los *completos*, al agregar una nueva placa a la serie *abreviada* (placas I, II, VI, VIII y X). La variabilidad de las cifras obtenidas, no se alteró notablemente, y los resultados *resumidos* únicamente dejaron de registrar aquellos resultados que estaban indicados en forma muy limitada en los registros *completos*.

Parte II

Se hizo la prueba de Rorschach a un grupo de 60 pacientes en el orden siguiente: Placas I, II, VI, VIII, X; IV, V, VII, III, IX. Las primeras 5 placas constituyeron la prueba *resumida*, cuyos resultados se cotejaron con los de la prueba *completa*. La correlación media para las principales categorías de clasificación fué de 0,93; para las categorías menos frecuentes, no fué tan satisfactoria, siendo el contenido intrínseco idéntico en los dos aspectos de la prueba. No se considera que esta simplificación pueda dar con certeza una idea exacta del modo de reaccionar del paciente, que se podría esperar si éste hubiera sido sometido a la prueba con las 10 placas, pero de todos modos se pueden obtener ciertos datos, teniendo en cuenta que, dentro de ciertos límites, ésta representa una prueba dentro de la otra prueba habitual.

RESUME

Partie I

Dans le but de vérifier si des inexactitudes importantes résulteraient de la simplification de l'essai Rorschach, on a extrait les notes pour quatre planches (I, II, VI & X) des 47 protocoles publiés par Beck. Les corrélations entre ces notes *abrégées* et les notes *completes* dont elles font partie s'échelonnent de +0,93 à +0,49. Lorsqu'on ajouta une planche de plus à la série *abrégée* (soit Planches I, II, VI, VIII & X), on obtint un plus grand degré de corrélation entre les notes *abrégées* et les notes *complètes*. On n'a pas observé de variation significative des notes enregistrées, et le système *abrégé* ne s'est montré insuffisant que pour les notes qui n'étaient que faiblement représentées dans le système *complet*.

Partie II

L'essai Rorschach a été appliqué à un groupe de 60 sujets dans l'ordre suivant—Planches I, II, VI, VIII, X; IV, V, VII, III, IX. Les cinq premières planches constituaient l'essai *abrégé*, qu'on mit en corrélation avec les résultats pour la série *complète*. La corrélation moyenne pour les principales catégories de notes a été de +0,93; les corrélations pour les catégories de notes moins fréquemment enregistrées ont été moins satisfaisantes; la teneur principale a été identique dans les deux aspects de l'essai. On ne prétend pas que cette forme d'essai abrégé puisse donner une indication exacte de la réaction à attendre du sujet pour la totalité des dix planches, mais un certain avantage peut découler du fait que dans certaines limites cette épreuve constitue un essai dans le cadre de l'essai complet.

ZUSAMMENFASSUNG

I. Teil

Um festzustellen, ob sich grundlegende Ungenauigkeiten ergeben, wenn man den Rorschachtest vereinfacht, wurden die Resultate von vier Karten (I, II, VI, und X) aus den 47 von Beck veröffentlichten Protokollen getrennt wiedergegeben. Die Korrelation dieser *gekürzten* Resultate mit den *gesamten*, denen sie angehörten, bewegte sich zwischen +0,93 und +0,49. Wenn eine weitere Karte der *gekürzten* Serie beigefügt wurde (d.h. Karten I, II, VI, VIII, und X), wurde eine höhere Korrelation zwischen *gekürzten* und *gesamten* Resultaten erzielt. Die

Variabilität der Resultate wurde nicht bedeutsam verändert, und in der *gekürzten* Serie fehlten nur diejenigen Resultate, die in der *gesamten* Serie nur schwach vertreten waren.

II. Teil

Der Rorschachtest wurde bei 60 Versuchspersonen in der folgenden Reihenfolge angewendet: Karten I, II, VI, VIII, X; IV, V, VII, III, IX. Die ersten fünf Karten stellten die *gekürzte* Serie dar, deren Resultate dann mit denen der *gesamten* Serie verglichen wurden. Die durchschnittliche Korrelation für die Hauptkategorien ergab sich als +0,93; die Korrelation für weniger häufig bewertete Kategorien war weniger zufriedenstellend; der Hauptinhalt war bei den beiden Serien identisch. Man kann nicht behaupten, dass diese Art der Kürzung ein absolut zuverlässiges Bild der von der Versuchsperson zu erwartenden Leistung für alle zehn Karten ergibt; es mag jedoch von Nutzen sein, dass die *gekürzte* Serie sozusagen eine Probe im Rahmen der grösseren Probe darstellt.

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